A COMPARATIVE ANALYSIS OF THE EFFICACY OF YELLOW LASER(577nm) AGAINST GREEN LASER(532nm) IN THE TREATMENT OF DIABETIC MACULAR EDEMA

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CHENNAI

CERTIFICATE

This is to certify that this dissertation entitled "**A COMPARATIVE ANALYSIS OF THE EFFICACY OF YELLOW LASER(577nm) AGAINST GREEN LASER(532nm) IN THE TREATMENT OF DIABETIC MACULAR EDEMA"** is a bonafide work done by Dr. Shreyas Ramamurthy under our guidance and supervision in the Retina and Vitreous Services of Aravind Eye Hospital and Post Graduate Institute of Ophthalmology, Madurai during the period of his post graduate training in Ophthalmology for May 2010 – April 2013.

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DECLARATION

I, Dr.Shreyas Ramamurthy, hereby declare that this dissertation entitled, "**A COMPARATIVE ANALYSIS OF THE EFFICACY OF YELLOW LASER(577nm) AGAINST GREEN LASER(532nm) IN THE TREATMENT OF DIABETIC MACULAR EDEMA"** , is being submitted in partial fulfilment for the award of M.S. in Ophthalmology Degree by the Tamil Nadu MGR Medical University in the examination to be held in April 2012.

I declare that this dissertation is my original work and has not formed the basis for the award of any other degree or diploma awarded to me previously.

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INTRODUCTION

Diabetes mellitus (DM) is a debilitating disease that over the last few decades has undergone a lot of research and a lot has been understood about the pathophysiology of the disease. Diabetic macular edema is the most common cause of visual loss in the diabetic population.

It was demonstrated in 1985 by the Early Treatment Diabetic Retinopathy Study (ETDRS) that focal (direct/grid) laser photocoagulation reduces moderate vision loss from diabetic macular edema (DME) by 50% or more. A number of studies along with the ETDRS have also shown the adverse effect which the lasers can have on the ocular tissue thus compromising the outcome of the laser treatment. Thus, many newer laser machines that work through varied mechanisms and wavelengths have been developed over recent years to reduce the rate of complications.

The variety of lasers available today has given an added dimension to the treatment of Diabetic Macular edema. Its imperative to strike a balance in a laser which will have the maximum effect and still cause minimal damage.

Our aim in this study is to evaluate the efficacy of the 577 nm laser, which is a yellow light laser, and compare it to the conventional 532 nm green laser that is widely used. The 577 nm yellow wavelength laser when compared to the 532nm green light has a higher affinity for oxyhemoglobin and a slightly lower affinity for melanin and almost no affinity for macular xanthophylls. Yellow light is also scattered very little during its transit through the ocular tissue and causes minimal photochemical reactions in the surrounding tissues.

Thus the major advantage in using the 577 nm yellow laser theoretically would be to reduce energy requirement and to obtain the same results as with green 532 nm. The reduced energy would lead to less retinal toxicity and damage due to reduced absorption by the xanthophylls. Our objective in this study would be to observe whether the theoretical advantage translates to a more effective treatment in reality.

HISTORICAL PERSPECTIVE

1946 – Meyer Schwickerath first started to investigate the creation of retinal burns using sunlight. [3]

1958 – Xenon arc lamp was introduced which emitted spectrum similar to sunlight and offered a longer life lamp with relatively high uniform output. [4]

1960 - Maiman introduced the first ophthalmic laser which used a ruby crystal. The ruby output had a wavelength of 694nm. Was highly coherent, allowing for better transmission through the ocular media with more precise retinal burns. [5]

Late 1960s – Argon blue laser revolutionised the field of ocular laser photocoagulation. The argon wavelength was relatively well absorbed by haemoglobin permitting direct closure of retinal vasculature. [6]

Early 1980s – Krypton red laser was introduced which penetrated through cataracts and haemorrhage extremely well and had negligible absorption by macular xanthophylls.[7]

Mid 1980s – Dye laser was developed & offered a continuous spectrum of wavelength from 560 – 640nm. The yellow (577nm) and red (640nm) wavelengths became the most clinically useful. Dye yellow emerged as

an ideal wavelength for closing vascular lesions, since it is near the peaks of the haemoglobin absorption curves. The benefits of dye red were virtually identical to those of Krypton red.[8]

Late 1980s - The development of the diode laser in the late 80s produced a compact, portable air cooled instrument with an output of 805-810nm. [9]

Early 1990s – Solid state, frequency doubled ND:YAG laser was first described in 1971, but did not become commercially available until early 1990s. The absorption characteristics of its wavelength, 532nm, most closely resemble those of dye yellow. [10]

Mid 2000s – The development and launch of the solid state yellow laser (577nm) which has recently become commercially available. It has similar advantages to that of the dye yellow laser and is a more stable substance making it a more viable product.

DIABETES MELLITUS

Diabetes mellitus is a major of metabolic disorder characterised by high blood sugar, due to insufficient production of insulin or due to poor responsiveness of the peripheral tissue to insulin. [12] The increased osmolarity of the blood due to the increased level of blood sugar causing the classical triad of symptoms of diabetes namely, polyuria (frequent urination), polydipsia (increased thirst) and polyphagia (increased hunger).

Diabetes Mellitus can be broadly divided into three major types:

a) Diabetes Mellitus Type 1: This results from the inability of the pancreatic tissue to produce insulin and therefore the patient requires exogenous insulin. Hence this condition is referred to as Insulin Dependent Diabetes Mellitus (IDDM).

b) Diabetes Mellitus Type 2: This condition is characterised by an increased peripheral resistance to the insulin produced. This increased resistance is maximal in the peripheral adipose tissue. As this disease can result even in the presence of normal levels of circulating insulin, it has been referred to as Non-Insulin Dependent Diabetes Mellitus (NIDDM)

c) Gestational Diabetes: It is characterized by the development of a high blood glucose level during pregnancy in a previously normal patient. It may however precede development of type 2 DM.

Insulin Dependent Diabetes Mellitus:

Type 1 diabetes mellitus by an immune mediated pathology is characterized by loss of beta cells in the pancreas which produce insulin. An autoimmune T cell mediated attack causes destruction of the Beta cells in the islets of the pancreas[16]. Unlike in type 2 diabetes, the sensitivity and responsiveness to insulin in the peripheral tissues is essentially normal. Type 1 diabetes usually affects the younger age group and onset occurs in childhood or early adulthood.

Non Insulin Dependent Diabetes Mellitus:

In Type 2 diabetes mellitus there is an increased peripheral insulin resistance, which may or may not be combined with relatively reduced insulin secretion [12]. The increased peripheral resistance of body tissues to insulin is believed to be due to defects or decrease in the amount of the insulin receptor. Type 2 diabetes usually presents only in adults and it is the most common type.

Signs and symptoms

The classical triad of symptoms of uncontrolled diabetes mellitus are weight loss, polyuria (frequent urination), polydipsia (increased thirst) and polyphagia(increased hunger). [22]. The origin and progression of signs and symptoms is rapid (weeks or months) in type 1 diabetes, where as they usually develop much more slowly and may be subtle or absent in type 2 diabetes.

Emergencies in Diabetes Mellitus:

A) Diabetic Ketoacidosis: It is most common in Type I Diabetics. There is a state of metabolic dysregulation which is characterized by the smell of acetone and a rapid, deep breathing known as Kussmaul breathing. This is accompanied by nausea, vomiting and abdominal pain, and altered states of consciousness.

B) Hyperosmolar Nonketotic Coma:

It is more common in type 2 diabetics though its incidence is rare. It is characterized by a very high blood sugar with a very high blood osmolarity resulting from the raised sugar levels. It is mainly caused due to the dehydration.

Long term Complications:

The risk of long term complications increases with the duration of the disease in all forms of diabetics and typically takes about 10-20 years for them to develop. Diabetic complications can be broadly divided in to:

a) Macrovascular complications: It is caused by the accelerated atherosclerotic changes in the large vessels that occurs in diabetes and can lead onto ischemic heart disease, stroke and peripheral vascular disease. There is a two fold increase in the risk of cardiovascular disease [23].

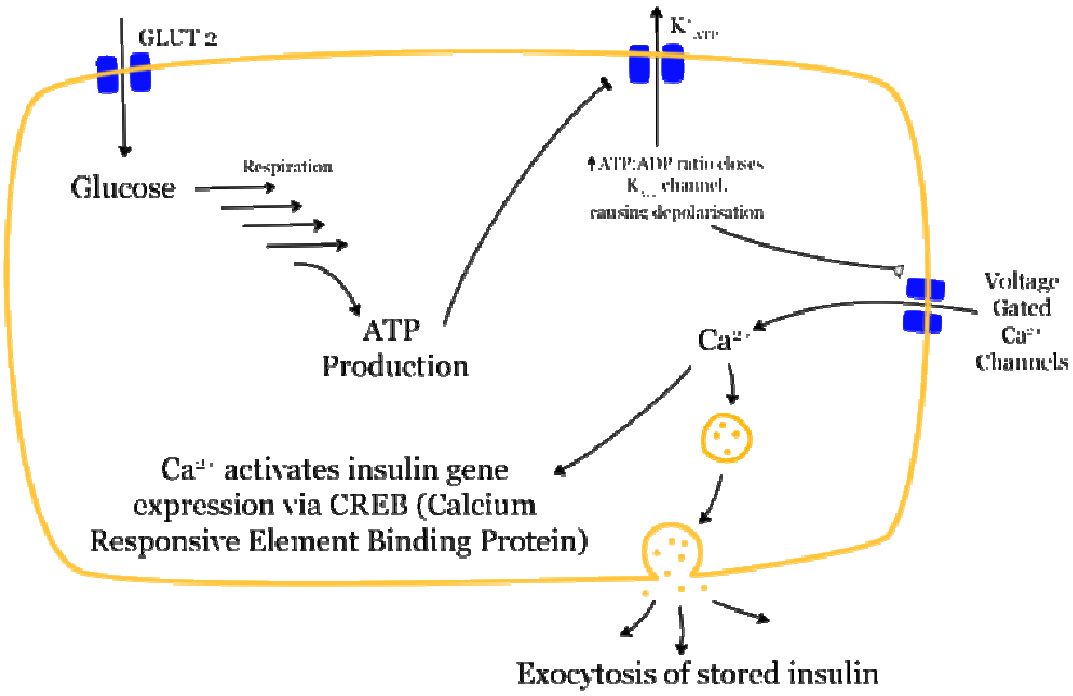
b) Microvascular complications: It results due to the damage that occurs in small blood vessels [24] and can lead onto diabetic retinopathy, diabetic nephropathy and diabetic neuropathy.

Pathogenesis:

The regulation and uptake of glucose from the blood stream into the tissues is mainly governed by Insulin. Hence either a lack of insulin or an increased insensitivity to it plays a central role in all forms of diabetes mellitus.

The carbohydrate content in the food is digested in the gut and is broken down into its most simple form the monosaccharide glucose which is the principal carbohydrate energy source used by the body. From the islets of

Langerhans in the pancreas the beta cells secrete Insulin which is released into the blood stream in response to rising levels of blood glucose usually after a meal. About two-thirds of the body's cells require insulin to absorb glucose from the blood for use as fuel, for conversion to other needed molecules, or for storage.



Insulin also plays a central role in the storage of glucose in the form of glycogen in liver and muscle cells. The release of insulin from Beta cells falls when there is a lowered blood glucose levels. The hormone glucagon acts in the reverse manner as insulin and is released from the alpha cells of pancreas. It cause breakdown of glycogen to glucose and enables

raising blood glucose levels. This usually occurs in the liver cells when the level of glucose is low in the blood stream.

Insulin plays a central role in other anabolic processes, such as cell growth and duplication, protein synthesis, and fat storage. The insulin levels either being high or low acts as an important trigger in controlling bidirectional processes of metabolism from a catabolic to an anabolic direction, and vice versa. In particular, a low insulin level is the trigger for entering or leaving ketosis (the fat-burning metabolic phase).

When insulin levels are insufficient or if there is insulin insensitivity or resistance then glucose will not be absorbed properly by those body cells that require it, nor will it be stored appropriately in the liver and muscles. The resultant effect is a persistently elevated levels of blood glucose and subsequently there is poor protein synthesis, and in increased fat breakdown resulting in ketosis and acidosis.

With the gradual increase of the glucose concentration in the blood is beyond the renal threshold that is above 10mmol/L, there is incomplete reabsorption of glucose in the proximal renal tubuli. This results in release of glucose in urine i.e. glycosuria. This causes increased osmotic pressure of the urine and decreased reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss. This increased fluid loss results in a dehydrated state that

triggers the central thirst mechanisms resulting in increased fluid intake (polydipsia).

Thus diabetes with its myriad presentation is a systemic disease which with its microvascular and macrovascular complications can cause increased morbidity and mortality if not effectively controlled.

DIAGNOSIS AND INVESTIGATIONS OF DIABETES MELLITUS

Diagnosis

Diagnosis of Diabetes [28, 29]			
	Fasting (mg/dl)	**Post Prandial (mg/dl)**	**HbA_{1c}**
Normal	<110	<140	<6.0
Impaired glucose tolerance	>110 but <126	>140 but <200	6.0–6.4
Diabetes mellitus	>126	>200	≥6.5

The criteria for diagnosing diabetes mellitus include the following:

1) Fasting blood sugar above 126mg%.
2) Post prandial blood sugar, that is the level of measured plasma glucose after 2 hours of consumption of 75g of glucose, which is greater than 200mg%.
3) The signs and symptoms of hyperglycemia accompanied by a raised blood glucose levels.
4) Glycated hemoglobin (Hb A1C) ≥ 6.5%(30)

It is preferable to measure a fasting glucose level because of the ease of measurement rather than the considerable time taken up by conducting the glucose tolerance testing as it takes two hours to complete and offers no significant prognostic advantage over the fasting test. [31] According to the current criteria two fasting glucose measurements above 126 mg/dl is considered diagnostic for diabetes mellitus.

Those with fasting glucose levels in the intermediate range from 110 to 125 mg/dl are grouped as impaired fasting glucose. [32]

Candidates with plasma glucose above 140 mg/dl but less than 200 mg/dl, which is measured two hours after a 75 g oral glucose are grouped as impaired glucose tolerance. [33].

Glycated hemoglobin is more sensitive predictor of risk of cardiovascular disease and death than fasting glucose. [34]

Glycated hemoglobin (HbA1c)

The content of average plasma glucose concentration over a period of time can be measured indirectly by the glycated haemoglobin. The glucose in the blood stream through a non-enyzmatic glyaction pathway gets incorporated into the haemoglobin chain. As the amount of blood glucose increases the glycated haemoglobin also increases. The level of glyacted haemoglobin lasts as long as the life span of the RBC containing

the haemoglobin. Therefore the glycated hemoglobin serves as a marker of the level of blood glucose over the last 3-4 months.

In 2010, American Diabetes Association Standards of Medical Care in Diabetes devised the additional criteria for diagnosis of diabetes being HbA1C of >6.5%. [25]

A high level of HBA1C also serves as a marker for cardiovascular disease, nephropathy, and retinopathy. Monitoring HbA_{1c} in diabetic patients may help avoid long term complications. [26]

DIABETIC MACULOPATHY

Among patients with diabetes the most common cause of visual impairment is Diabetic macular edema. The pathogenesis of DME is complex and multifactorial. It occurs mainly as a result of disruption of the blood--retinal barrier, which leads to increased accumulation of fluid within the intraretinal layers of the macula. [38,43,87]

A. CLINICAL DESCRIPTION AND CLASSIFICATION

Diabetic macular edema is diagnosed as retinal thickening in the macula using Fundus slit lamp biomicroscopy. The Early Treatment Diabetic Retinopathy Study (ETDRS) defined DME as retinal thickening or presence of hard exudates within 1 disk diameter of the centre of the macula. This definition has been used consistently in most of the diabetes related research studies. [51,71,72] To characterize the severity of macular edema and for treatment guidelines, the term clinically significant macular edema (CSME) is used.

Macular edema is clinically significant if one of the following conditions is present:

retinal thickening within 500 microns of the centre of the macula;

(or)

hard exudates within 500 microns of the centre of the macula if associated with thickening of the adjacent retina;

(or)

a zone of retinal thickening of atleast one disk area in size, at least part of which is within 1 disk diameter of the centre of the macula. [51,71]

B. FOCAL VERSUS DIFFUSE DIABETIC MACULAR EDEMA

Clinically significant macular edema is further classified into focal or diffuse, depending on the leakage pattern seen on the fluorescein angiogram (FA). Leakage noted on FA is not synonymous with edema or thickening. The FA is used to identify areas of increased vasopermeability, for example, leaking microaneurysms or capillary beds, and to evaluate retinal ischemia. Leakage on the angiogram does not necessarily indicate retinal edema since extracellular edema requires that the rate of fluid ingress into the retina (i.e., as indicated by leakage on the FA) exceed the rate of fluid clearance from the retina (e.g., via the RPE pump).

In focal CSME, [42] multiple microaneurysms produce focal points of retinal hyperfluorescence on FFA.[39,238]. The discrete leaking microaneurysms are thought to cause retinal thickening. Commonly, these leaking microaneurysms are surrounded by circinate rings of hard

exudates. The exudates are lipoprotein deposits in the outer retinal layers. [40] In diffuse DME, there is a diffuse hyperpermeability response of the retinal capillaries or intraretinal microvascular abnormalities within the macula that produce areas of diffuse leakage are noted on the FA. [39,52] There may be a generalised breakdown of the inner blood retinal barrier which can be associated with cystoids edema with fluid accumulation in the outer plexiform layer. [41,68]

Focal DME is characterized by well-defined, discrete areas of leakage from the microaneurysms on the FA, in comparison to diffuse DME, which is characterized by generalized areas of leakage in the area centralis. Furthermore, focal DME is responsive to focal laser photocoagulation, whereas diffuse DME represents a more challenging clinical situation and is refractory to laser photocoagulation in many cases.[54,58,74.] Grid pattern of laser treatment may be helpful in certain cases. Compared to those with less severe non-proliferative diabetic retinopathy, the relative risk for diffuse macular edema is 6.2 times greater in patients with very severe nonproliferative diabetic retinopathy and 7.7 times greater in patients with proliferative diabetic retinopathy. [53]

C. EPIDEMIOLOGY

In one study, the incidence of DME over a 10-year period was 20.1% among patients diagnosed before age 30 years (younger onset) and 39.3% among patients diagnosed after age 30 (older onset). [191] Diabetic macular edema, in this study, was defined as thickening of retina within 1 disk diameter of centre of the macula. The Diabetes Control and Complications Trial (DCCT) reported that 27% of patients develop macular edema within 9 years of diabetes onset. [37,61,71,72,100] . The frequency of DME increases with the duration and the severity of diabetes. [70,80] . Older onset diabetic patients have a tendency to develop macular edema earlier in the course of their disease (prevalence: 3-8% with up to 3 years of disease duration) compared to younger onset diabetic patients (prevalence: 0.5% with up to 10 years of disease duration)

PATHOGENESIS

A. BLOOD--RETINAL BARRIER

The common pathway that results in DME is disruption of the BRB [49]. The BRB compartmentalizes the neurosensory retina from the vascular component of the eye. Maintenance and proper functioning of the BRB is a complex process and involves the interaction of several factors.

The BRB consists of two major components: the outer barrier and the inner barrier. The inner BRB is a biological unit formed primarily by tight junctional complexes between retinal vascular endothelial (RVE) cells and a well-differentiated network of glial cells (astrocytes and Muller cells) that operates to maintain a low permeability environment; [86] the outer BRB is formed by tight junctions between retinal pigment epithelium (RPE) cells and includes zonula occludens with prominent desmosomes. [45,48]

1. Inner Blood--Retinal Barrier

The inner BRB comprises capillary endothelial cells with intercellular tight junctions within a closely differentiated network of neurons and glial cells. [59,86,96]. The tight junctions include zona occludens, which serve as one of the most important elements in regulating BRB. Several cells assist in maintaining the inner RB as mentioned subsequently.

a. Glial Cells

Astrocytes guide the migration of retinal vessels during fetal life and, in combination with Mueller cells, induce the formation of barrier properties and tight junction proteins. [60,96,98] . The astrocyte foot processes very tightly surround the retinal vascular endothelial cells creating a

microenvironment of BRB. Structural and functional changes are noted in neurons and glial tissue of the retina in early diabetes, much before changes in the retinal vasculature are noted. [40,76,78,84]

b. Pericytes

Pericytes are microvascular mural cells that provide vascular stability [62] . Pericytes play an important role in controlling the inner BRB. Morphological changes of pericytes are seen in early diabetes; the cells become rounder, with reduction in the number of processes, and develop impaired cell adhesion to the underlying matrix [50]. Apotosis of retinal capillary pericytes occurs during early diabetic retinopathy. [77]. Loss of pericytes ensues and leads to the weakening of the pericyte sheath, outpouching of capillaries, microaneurysms, and breakdown of the inner BRB. [47,50,73,93,99]. Loss of pericytes in early diabetic retinopathy may be related to the retinal capillary endothelial death that leads to capillary dilation, microaneurysms, retinal ischemia, production of VEGF, increased vascular permeability, and angiogensis.

C. Vascular Endothelium

Endothelial cell death is a hallmark of diabetic retinopathy. It has been shown that endothelial cell death precedes the formation of acellular

capillaries, which progresses over time. The resultant acellular capillaries lead to irreversible retinal ischemia. The exact mechanism that initiates this cascade of events in diabetic eyes is still largely unknown. Abnormal adhesion of leukocytes to the diabetic vascular endothelium is present early in the disease and is considered an important inciting factor in animal models of diabetic retinopathy, which is significantly associated with retinal capillary occlusion and breakdown in BRB. [64,65]

D. Retinal Vascular Leukostasis

Although diabetes is not traditionally considered an inflammatory disease, increased numbers of leukocytes are noted in the retinal vasculature. [67] Leukocyte adhesion to the capillary endothelium is one of the first histological changes seen in diabetes. [64,82,91]. Leukostasis within the retinal vasculature may play a crucial role in the endothelial cells death. Leukocyte adhesion to the diabetic retinal vessel wall causes apoptosis of pericytes and endothelial cells, vascular obstruction, subsequent non-perfusion, and release of cytokines that increase vascular permeability. [67,82]

e. Advanced Glycation End-Products (AGEs)

Chronic hyperglycemia leads to the formation and accumulation of AGEs that may be a primary contributor to diabetic microvasculopathy. AGEs form on the amino groups of proteins, lipids, and DNA with complex cross-links and lead to modification in the structure and function of proteins. [64]

Advanced glycation end-products activate ICAM-1 in the endothelial cells, possibly by increasing the levels of transcription factors such as NF-kappa B. [65] There is indirect evidence to show a strong correlation of AGE products with leukostasis. Retinal leukostasis and disruption of the BRB has been shown to occur after the infusion of AGE in normal mice [82]. Increased expression of ICAM-1 and CD-18 parallels leukocyte adhesion. Specific inhibition of ICAM-1 or CD18 prevents leukocyte adhesion and breakdown of the BRB. [64]. Advanced glycation end-products are found in vitreous, especially at the vitreoretinal interface on the posterior vitreous cortex and the internal limiting membrane (ILM). Their receptors, RAGEs, are attached to the footplates of the Muller cells spanning ILM and the external limiting membrane.

F. ROLE OF VASOACTIVE FACTORS

Several vasoactive factors (e.g., VEGF, protein kinase C [PKC], heparin, angiotensin II, PEDF, metalloproteases) and biochemical pathways may be affected by sustained hyperglycemia in diabetes, which may influence the development of structural and functional changes in diabetic retinopathy [44,56,90,100]. Increased levels of histamine seen in diabetic patients increases vasopermeability directly and also indirectly by upregulating PKC.

VEGF is increased in the retina of diabetic rats as well as in diabetic humans. VEGF is produced by RPE cells, ganglion cells, Muller cells, pericytes, endothelial cells, glial cells, neurons, [55,95] and smooth muscle cells of the diabetic retina. Upregulation of VEGF by hypoxia occurs in all of these cell types [94,95]. Muller cells are the most important source of VEGF in the retina due to their high rate of glycolysis. VEGF produces conformational changes in the tight junctions of retinal vascular endothelial cells [38]. VEGF induces phosphorylation of the tight junction proteins, occludin and ZO-1, which leads to increased vascular permeability by phosphorylation of adherent junction and cytoskeletal proteins of vascular endothelial cells and induction of fenestrations in endothelial cell membranes. [90] Other cytokines, such as insulin-like

growth factor-1 (IGF-1), interleukin-6 (IL-6), and PKC-beta, also promote the expression of VEGF and BRB disruption. Hyperglycemia itself can enhance the response of retinal endothelial cells to IGF-1.[81] IGF-1 can regulate the expression of VEGF in RPE cells. A positive correlation is observed between the serum or vitreous concentration of IGF-1 and extent of neovascularization in diabetic retinopathy. Insulin-like growth factors (IGF-1 and IGF-2) are upregulated in vitreous of patients with ischemic retina. [88]

VEGF also may be associated with the early inflammatory changes seen with diabetic retinopathy and DME. In early diabetes, vitreous levels of VEGF are elevated. In rodents, increased levels of VEGF are associated with upregulation of ICAM-1 leading to retinal leukostasis and endothelial cell damage. [64,66,89]

C. VITREORETINAL INTERFACE

Clinical and anatomical evidence indicates that abnormalities in the structure of the vitreoretinal interface may play an important role in the pathogenesis of DME. [63,75,97] DME may be exacerbated due to persistent vitreomacular traction by the residual cortical vitreous on the macula after PVD, thickened and taut posterior hyaloid that may or may not be

adherent to ILM, macular traction due to tractional proliferative membranes, or loculation of cytokines in the pre-macular vitreous pocket. A diabetic retina compromised due to microvascular abnormalities may be vulnerable to increased exudation in the presence of any macular traction.

OPTICAL COHERENCE TOMOGRAPHY

OCT can be used to generate cross-sectional or three-dimensional images by measuring the echo time delay and magnitude of back-reflected light. Quantitative measurements of retinal thickness and volume can be done by easy non invasive modality. The quality of the scans and images obtained by OCT can only compared with histology specimens and is far superior to any other non invasive diagnostic technique. [103,104,105,106]

Since it was first described by Huang et al in 1991[107] OCT has grown leaps and bounds. The retina was first demonstrated in vivo via imaging in 1993 [108], the first commercially available OCT device, 'Zeiss OCT,' was developed in 1996. The axial resolution was 10mm and it had a speed of 100 A-scans per second. It was only in 2002 when the Zeiss Stratus OCT became available when OCT found a more widely accepted place in clinical ophthalmology. The increased speed of image acquisition by the Stratus OCT0, 400 A-scans per second, resulted in improved image quality. The improved quality of images along with widespread availability of clinical data showed the utility of OCT in daily ophthalmic practice. Though not yet available on a large scale Ultra high resolution OCT is capable of an axial resolution of 2– 3mm and can provide subcellular detail. [103].

Time domain detection is used in Stratus OCT in which the reference mirror position and delay require mechanical scanning to produce A-scans .A high speed spectrometer that measures light echoes from all time delays simultaneously in the spectral or Fourier-domain OCT, has significant advantages over time-domain detection. The reference mirror need not be mechanically scanned in Spectral-domain OCT and it also has improved sensitivity and allows for faster sampling speed and improved signal-to-noise ratio. Spectral domain OCT available today have achieved image acquisition speeds greater than 20 000 A-scans per second and axial resolutions of 4–7mm. [109,110]

The second inner hyper reflective band is used as the outer border of the retina in the SOCT Copernics and RTVue-100. The Cirrus HD-OCT & Spectralis OCT identify the most outer hyperreflective band as the outer border of the retina. These differences were detected by comparing the measurement of retinal thickness on the same retinas and by this Stratus OCT measured the lowest central retinal thickness whereas Spectralis OCT and Cirrus HD-OCT measured the highest.

The best intersession repeatability was found in the Spectralis OCT [110]. These differences are of clinical importance if the same patient undergoes sequential scans on tomograms from different platforms.

OCT enables us to detect and rapidly quantify macular edema [112]. In SD-OCT the pictures are depicted in gray scale and not in colour scale but it helps accurate detection of more subtle differences in tissue reflectivity. [114].

Terms used in OCT:

(1) Retinal thickness: distance between the RPE and the ILM in microns excluding the subretinal fluid.

(2) Retinal thickening: calculated value equal to the retinal thickness minus the population mean for the variable under consideration.

(3) Central subfield: circular area 1mm in diameter centered on the centre point.

(4) Centre point: calculated by point of intersection of the radial scans.

(5) Centre point thickness: average of the thickness values of the radial scans.

(6) Central subfield mean thickness: mean value of the thickness values obtained within the central subfield.

Assessment of Visual acuity by OCT:

A good correlation has been found between the central retinal thickness measured by OCT and visual acuity in many studies. [102,115,120] . As deduced from these studies Central retinal thickness measurement by

OCT has a more significant effect on visual acuity than does age or other parameters affected visual acuity like fluorescein leakage, perifoveal capillary blood flow velocity or perifoveal capillary occlusion [102,118] The DRCR.net displayed a lot of variability in results in a large study as they found a paradoxical improvement in vision occurred with increased thickening in 1–17%, and a paradoxical worsening of vision occurred with decreased thickening in 18–26%. These variations were probably due to the fact that The DRCR.net study [102] did not analyze the duration of existing edema at baseline nor did it have complete data regarding the degree of macular ischemia which could cause an alteration in the macular edema and its correlation with central visual acuity.

Optical coherence tomography patterns in diabetic macular edema:

OCT has provided us an excellent qualitative and quantitative method to classify diabetic macular edema. [120,121,126]

The types are:

a) Diffuse spongiform macular edema

b) Cystoid macular edema

c) Serous retinal detachment

d) Taut posterior hyaloids

Diffuse retinal thickening is described as a sponge like swelling or edema of the retina with a generalized & heterogeneous image with mild hyporeflectivity. Some authors suggest that there must be a quantitative increase in the central subfield mean thickness with a value like 200 microns on Stratus OCT. The threshold may vary according to the platform being used. Diffuse retinal thickening is the commonest reported finding via OCT in eyes with DME and are present in 88-100% of eyes with DME. Diffuse retinal thickening alone without any other characteristics patterns described by OCT is present in 36–42% [120,122,127].

Cystoid macular edema is described by the presence of intraretinal, round or oval cystoid areas of low reflectivity that were separated by septae which are highly reflective. This reflectivity with the cystoids spaces in DME is has greater reflectivity than the ones found in cystoids spaces associated with Type 2 IJT according to Barthelmes et al. Due to the exudative nature of DME with breakdown of the inner bloodretinal barrier and therefore a higher concentration of protein may be the reason behind the increased intrinsic reflectivity in diabetic cystoid edema.. Cystoid macular edema is found through OCT in 44–47% of eyes with DME [120,122,127].

Sub retinal detachments have been described on OCT as a focal, arch-like elevation of neurosensory retina overlying an accumulation of subretinal fluid which is hyporeflective and the resultant space is dome shaped. The overlying posterior border of the retina is typically hyperreflective and thick due to an accumulation of unphagocytosed photoreceptor outer segments. SRDs under the fovea have been reported in 3–31% of patients with DME. [102.126.127.128.129].

Vitreo Macular Interface Abnormalities (VMIAs) include the presence of epiretinal membranes, vitreomacular traction or both. An epiretinal membrane is visible in OCT as a thickened hyperreflective band along the inner aspect and may have the pseudohole which will appear as an incomplete or partial defect in the membrance. Vitreomacular traction on the other hand was identified by a hyperreflective band that is in apposition with the inner surface of the retina at a particular site(s) but is elevated above the surface of the retina elsewhere unlike a epiretinal membrane which is seen apposed right through [127,130]. VMIAs appear to occur in 14–16% of eyes with DME [122,127]. The role of VMIAs in recurrent or persistent macular edema has been better understood only after the advent of OCT.

OCT has now become invaluable in the qualitative and quantitative assessment of DME. It helps to diagnose, prognosticate and also aids in delivering a targeted therapeutic approach to the various patterns of Diabetic Macular Edema.

FLUORESCEIN ANGIOGRAPHY

Fluorescein angiography is a gold standard method for identifying site or sites of leakage in Diabetic Macular edmea. [130,131] The leaking areas not only help us to identify the microaneurysms but targeted laser therapy can be given to them in the presence of an FA. Areas of non perfusion helps us to identify ischemic maculopathy and prognosticate and avoids unnecessary laser therapy.

Kang and coworkers categorized fluorescein angiographic leakage in DME into three different types:

1) focal leakage: well-defined focal area of leakage from microaneurysms or dilated Capillaries

2) diffuse leakage: It is believed to be a diffuse hyperpermeabilty response of the macular capillary bed

3) diffuse cystoid leakage: diffuse leakage and pooling of dye in the cystoid spaces that occur in DME in the late phase of the angiogram and can be correlated with OCT.[132]

The various patterns described above which can be elicited by FFA are very essential as they guide the mode of laser therapy that is to be administered. Focal leaks are treated with focal laser where as diffuse leaks are treated with grid lasers leaving a distance of 500 microns from the centre of the macula. [133].

OCT and FFA have therefore become essential prerequisites in the armamentarium of investigation of the retina for diagnosing and treating diabetic macular edema.

LASER PHYSICS

Laser is an acronym for "Light Amplification by Stimulated Emission of Radiation". Substances have the property to lase i.e. to absorb energy in uniform (either thermal, mechanical, electrical or light – all ophthalmic lasers presently use light as the stimulating source) and to emit a new form of light energy which is more useful. A substance which has the ability to lase possesses the unique property of transferring the electrons from the orbital to a second meta stable, spaced by a definite energy interval. Most electrons have been energised sufficiently by the stimulation source, so that they are in a meta stable orbit of higher energy. This sudden jump to a lower energy level causes the emission of a new form of light energy.

BASIC REQUIREMENTS FOR A LASER

A lasing medium is placed in a optical cavity that acts as resonator. The resonator cavity consists of two mirrors carefully aligned to each other; the laser material is in between them. One of the mirrors is partially transmitting so that some of the radiation will be coupled out of the cavity. Once the population inversion has been achieved, the laser radiation build up begins with some of the excited atoms in the medium emitting spontaneous radiation in various random directions. Some of the

radiation however, will hit one of the mirrors and return passing through the medium. The radiation is re enacting in exactly the correct wavelength (since the material itself radiated it) and will therefore stimulate some of the atoms into emitting radiation, thus amplifying the beam. After the wave bounces back from the other mirror, it will be amplified as it passes through the medium again. Consequently the device emits a highly coherent beam because of the geometry of the cavity.

PROPERTIES OF LASER ENERGY:

Light emitted by a laser differs from normal 'white' light in the following ways.

COHERENCE:

Like the photons in a light bulb which are emitted randomly, the resonator effect of the laser cavity causes the photons to be synchronised (i.e. in phase with each other in time and space.)

COLLIMATION:

Since light amplification occurs only for photons that are aligned with the mirrors, a nearly parallel beam is produced as opposed to the diverging beam of an incandescent lamp. Although limited divergence occurs with all laser beams, it is minimal enough to provide a small focal spot when the light is delivered through an optical system.

MONOCHROMACY:

Since the photons are emitted as a result of the release of energy between two defined levels of the atom, the resulting light has a narrow range of wavelengths.

HIGH INTENSITY:

The light amplification of a laser can produce a beam with significantly more intensity than the sun.

LASER INDUCED TISSUE EFFECTS:

Laser can have various target tissue depending on the wavelength and power used.

1. PHOTOCHEMICAL EFFECTS:

Ultraviolet and visible light absorption induces the formation or destruction of chemical bonds. [134]

2. THERMAL EFFECTS:

Most commonly encountered with retinal photocoagulation. Occurs when the visible or infrared light is absorbed by tissue pigments.[135]. Absorption of laser energy results in a 10 to 20 degree temperature rise which photocoagulates, or denatures, the absorbing tissue. The temperature rise in the fundus is highly localised, limited to within 1mm of the burns

centre. [136]. Photocoagulation intensity is directly proportional to the magnitude and duration of the temperature rise. Protein denaturation is seen clinically as tissue whitening.

Absorption Characteristics of lasers of different wavelengths:

MAJOR FUNDUS PIGMENTS	XANTHOPHYLLS	HEMOGLOBIN	MELANIN
ARGON BLUE GREEN (488nm)	++++	+++	+++
ARGON GREEN (514NM)	---------	+++	++++
Nd:YAG (532nm)	------	++++	++++
DYE YELLOW (577nm)	------	++++	++++
DYE RED (630nm)	------	+	+++
KRYPTON RED (647nm)	-------	+	+++
DIODE (810nm)	--------	-----	++

Most of the thermal damage created during posterior segment photocoagulation is achieved by the absorption of laser energy by melanin in the retinal pigment epithelium and choroid.

3. VAPOURISATION:

Possible with visible or infrared laser energy. There is micro explosion, when the temperature of water rises above the boiling point, as with carbon dioxide laser or following an overlay intense argon burn.

4. OPTICAL BREAK DOWN:

Occurs with infrared light of the Nd:YAG laser. An extremely high power results in plasma formation where electrons are stripped away from atoms and molecules. This leads to a shock wave that physically disrupts tissue. The desired disruption is independent of tissue pigmentation

LASER PHOTOCOAGULATION FOR DIABETIC MACULOPATHY

The value of laser photocoagulation for the treatment of diabetic macular edema has been demonstrated in a multitude of studies.

[3,5,2,7,4,13,7,13,8,14,1,14,2,14,9,15,0,15,1,15,2,15,3,15,4,15,5,15,7,15,8,16,0,16,8].

1. The Early Treatment Diabetic Retinopathy Study (ETDRS) Results

The ETDRS study was a prospective, randomized, multicenter clinical trial. It was designed to evaluate the effects of argon laser photocoagulation for macular edema. An improvement in the visual acuity of 16% was noted among the eyes with mild to moderate non-proliferative diabetic retinopathy with macular edema. The visual acuity remained unchanged in 77%, and worsened in 7% of treated eyes. After 2 years of follow up, there was a decline in the visual acuity with improvement being in only 11%, and it remained unchanged in 73% with worsening in 16% of untreated eyes after 2 years of follow-up. At 3 years of follow-up, the worsening of vision was only 12% in the treated group compared to 24% in the untreated eyes. If macular edema involved the center of the macula (vs macula-threatening edema), then the visual prognosis was worse, and the magnitude of the treatment benefit was greater. Specifically, the risk of moderate visual loss among patients with

center involving macular edema assigned to deferral was 32% at 3 years vs 15% in the treatment group. Of the 350 eyes with CSME, which had retinal thickening involving the center of the macula, at 1 year of follow-up, only 35% of eyes assigned to immediate photocoagulation had retinal thickening in the center of the macula compared to 63% of eyes assigned to deferred photocoagulation. The benefit of laser photocoagulation was statistically significant in these eyes. [51]

The risk of severe visual loss was not significantly different between the treatment and control groups in each category of retinopathy (i.e., those with less severe vs more severe retinopathy) using the previously described treatment criteria. At one year follow up, the risk of moderate visual loss was significantly lower in the treatment group compared to the control group. [145] Though risk of severe visual loss at 5 years follow up was lower in the treated group as compared to the control group, it was not evident whether the visual loss would be influenced by the timing of focal photocoagulation. [145]

It was concluded by the ETDRS investigators that early focal photocoagulation should be considered in all eyes with CSME. The treatment was associated with a lower risk of moderate visual loss, an increased chance of visual improvement, less loss of colour vision, and only minor visual field changes. [42,51,52,145]

The anatomical and functional success of eyes with diffuse DME treated with grid laser photocoagulation is not good. Even with multiple treatments, diffuse DME can be refractory to macular photocoagulation.[140,154] . Lee and Olk[154] reported limited visual improvement after grid laser in their large study of 302 eyes with diffuse DME with or without CME. At 3-year follow-up, some vision improvement was noted only in 15%; visual acuity was unchanged in 61%, and moderate loss of visual acuity occurred in 24% of eyes. Complications can be seen with laser photocoagulation. With time, RPE atrophy associated with the laser scars occasionally progresses under the fovea causing decreased vision. Also, subretinal fibrosis can develop and cause visual loss. [148,161,162]. Thus, grid treatment has limited efficacy for diffuse DME.

Alternative treatments such as intravitreal triamcinolone acetonide (IVTA) injections, intravitreal anti-VEGF drugs, and pars plana vitrectomy (PPV) in the presence of vitreomacular traction can be tried.

2. Mechanism of Action of Laser Photocoagulation

The exact mechanism of action of laser photocoagulation- induced resolution of DME remains to be unidentified though several hypotheses have been put forward. One explanation involves laser-induced

destruction of oxygen-consuming photoreceptors. Laser photocoagulation creates an increase in tissue temperature of 10°C, with heat spreading to adjacent RPE cells, photoreceptors, and choriocapillaries. Cell death and scarring (involving gliosis and RPE hyperplasia) occurs subsequently. The loss of the metabolically active outer tissue enables easier diffusion of oxygen from the choriocapillaries into the inner retina. [164,172]. Several investigators have demonstrated increased preretinal oxygen partial pressure in areas of laser photocoagulation, which Perry and Risco explained by the presence of the microvascular repair in the treated zones. [159,165,166] However, Wilson and Green demonstrated choriocapillaris loss in areas of argon laser photocoagulation and proposed that increased oxygen tension is unlikely to be found in those areas. [171]. Also, Wilson and coworkers have demonstrated a reduction of the retinal capillary area in the zone of laser photocoagulation and proposed that if the total area of the abnormal leaking vessels was reduced, the amount of leakage would be reduced, which would result in the resolution of the macular edema (for a given capillary permeability and hydrostatic pressure). [170]

Gottfredsdottir and coworkers studied the diameter of retinal arterioles, venules, and their macular branches before and after macular laser photocoagulation in eyes with DME. [144] The macular arteriolar branches

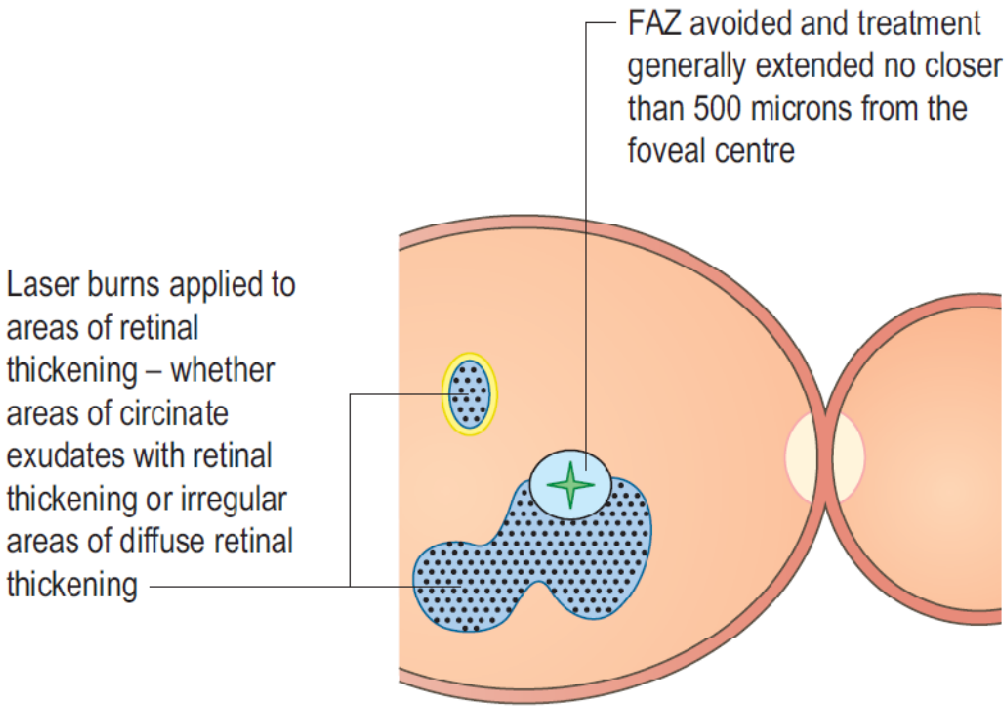
constricted by 20.2% ($p < 0.001$), and the venular branches constricted by 13.8%. The hypothesis put forward was that the improved retinal oxygenation post laser may lead to an autoregulatory vasoconstriction, which may improve DME.[144]

Another theory proposes that the beneficial effect of laser photocoagulation is due to restoration of a new RPE barrier. [41] The response of the RPE cells to the injury could be one of the following: in relatively small defects (<125 mm), the RPE defect can be filled by cell spreading; in larger defects, the cells can proliferate to resurface the area, and the RPE can produce cytokines (e.g., TGF-b) that antagonize the permeabilizing effects of VEGF. [134,143,156,173]. Several complications such as full-thickness retinal break, choroidal neovascularization, subretinal fibrosis, or symptomatic scotomata have been associated with higher energy levels of laser used in the treatment of diabetic macular edema. [146,147,169]. These complications can cause symptomatic visual loss.

Laboratory experiments have shown that RPE defects developing after selective subthreshold laser burns, sparing the neural retina but damaging RPE cells, are resurfaced by a new population of RPE cells.

Laser treatment for maculopathy is generally applied to areas of vascular leakage in one of the following patterns:

- **Focal** — This is used for circumscribed small areas of macular oedema where laser burns are applied to areas of microaneurysms and microvascular lesions in the centre of rings of hard exudates. Burns are commonly 100 microns in diameter with 0.08–0.1 second duration. Longer burn duration causes bigger burns and increased inner retinal damage by thermal spread, increased risk of bruchs membrane fractures, risk of haemorrhage and later choroidal neovascularization. The power is measured in milliwatts and should be kept as low as possible, typically starting at around 100 mW. Light burns are therefore used so that the laser reaction is just visible as a faint greyish-white reaction — a threshold burn. Generally no attempt is made to close retinal blood vessels.



Diagrammatic representation of focal/modified grid laser.

Management of diabetic retinopathy and diabetic maculopathy

● **Grid** — This is used when there is generalized diffuse macular oedema with foveolar involvement, typically using a spot size of 100–200 microns with a grid pattern of 100–200 burns of 0.08–1.0 s duration around the macula.The burns should be of light intensity and placed approximately one burn-width apart. If treatment is taken into areas with less macular oedema, then the power setting should be reduced. Treatment closer than 500 microns from the FAZ should be undertaken with caution but is occasionally done as repeat treatment in non-

responsive cases with persistent central oedema. The papillomacular bundle area can be safely treated provided there is macular oedema within this area, as in this situation the nerve fibre layer will be separated from the area of maximum energy uptake within the RPE and thus protected from thermal damage. Care should therefore be taken in treating 'dry' retina within this area and in re-treatments.

- **Modified grid** is similar to grid settings except the pattern is concentrated in a particular sector or sectors of retinal thickening rather than a general pattern around the foveal centre. In practice this pattern of treatment rather than grid laser is more commonly applied.
- Treatment was also given in the EDTRS to areas of eccentric capillary non-perfusion associated with retinal thickening in the macular area.

Complications of laser treatment

Para central scotomata are common after treatment close to the foveal centre. Patients should be warned that they may notice grey scotomata around their central vision for a few days to weeks after treatment. These usually however fade with time, but not always.

Transient worsening of vision can occur due to a transient increase in oedema following treatment; again this usually improves.

Choroidal neovascularization can occur and patients should be specifically warned of this complication if they have existing age-related macular degeneration (AMD). In this situation the choroidal neovascular membrane (CNVM) can actually be just part of the AMD natural history and not part of the laser treatment. High intensity small burns should be avoided which can rupture bruchs membrane increasing the likelihood of choroidal neovascularization. Also it should be noted that CNVM can occur spontaneously in patients with severe diabetic macular edema (DME).

Sub retinal fibrosis [146,147] can occur, but again this can occur in diffusely oedematous maculae as part of the diabetic maculopathy disease course. Similarly **epiretinal membrane** formation can occur. [161]

Laser scar expansion can occur, especially in myopic patients. [162]

Exudate deposition at the foveal centre can occur in extremely edematous retinae after treatment, and large exudates close to the fovea are also a risk factor. In patients with grossly oedematous maculae, or exudates very close to fixation, the option of fractionating the maculopathy treatment in a couple of sessions should be considered to try and avoid the risk of sub-foveal exudate deposition.

Increased ischaemia can occur rarely after maculopathy treatment, especially in patients with mixed maculopathy. If ischaemia is suspected an angiogram should be arranged so that this complication can be discussed with the patient if they wish. This is best undertaken with the patient looking at the angiogram with the operating clinician.

Colour vision and contrast sensitivity can be reduced following laser and again should be discussed if this is relevant to the patient's profession or hobbies in particular.

Focal laser treatment for diabetic maculopathy clearly works and the commonest cause for reduction in vision following laser treatment is actually progression in maculopathy rather than deterioration in vision secondary to laser.

REVIEW OF LITERATURE

- **Clin Experiment Ophthalmol. 2007 Sep-Oct;35(7):640-4.**

Subthreshold micropulse diode laser photocoagulation for clinically significant diabetic macular oedema: a three-year follow up.

Sivaprasad et al conducted a 3 year follow up study to report the efficacy of micropulse diode laser of 810nm wavelength for CSME. Their study had a total of 25 eyes from 19 patients. They showed an improvement in the visual acuity by 84% in the first year of follow up which improved to 92% by the third year. Although their final outcome was good they had 28% of cases showing recurrent CSME in the third year. This was among the longest follow up studies demonstrating the efficacy of the laser in treating CSME.

- **Ophthalmology. 1999 Feb;106(2):243-8.**

Treatment of diabetic macular edema: a comparison between argon and dye lasers.

Karacorlu et al performed a study comparing dye yellow and argon green laser in 85 eyes of CSME. They showed eighty five percent improvement in the argon laser group whereas there was eighty nine percent improvement in the dye yellow group. However this difference was not

statistically different. There was no difference both in terms of visual acuity as well as the amount of reduction of macular edema.

- **Comparative study of efficacy of focal photocoagulation in diabetic macular edema according to the wave length used**

Fernandez et al compared three different laser namely the blue green argon, the monochromatic green argon laser and the yellow dye laser with a total of 60 patients who were randomly assigned with twenty patients in each group. They reported a statistically significant difference in the dye yellow laser group in terms of visual acuity. The rate at which the exudates were absorbed were however similar in all three groups.

- **Br J Ophthalmol. 2009 Oct;93(10):1341-4.**

Prospective randomised controlled trial comparing sub-threshold micropulse diode laser photocoagulation and conventional green laser for clinically significant diabetic macular oedema.

Figueira et al in a ransomized controlled trial compared subthreshold micropulse diode against the green laser in eighty four eyes. They noticed no statistically significant difference in all parameters. The parameters

included visual acuity, contrast sensitivity and retinal thickness. Thus they concluded that both lasers had similar efficacy in the treatment of CSME.

- **Klin Monbl Augenheilkd. 2004 Jan;221(1):48-51.**

Grid laser photocoagulation in diffuse diabetic macular edema

Degenring et al evaluated the results of grid photocoagulation in forty one eyes of CSME with an average follow up period of 31 weeks. Of the forty one eyes they showed five eyes which gained visual acuity. Among the others twenty three remained stable whereas 13 lost vision by one line or more. They were one of the few studies which actually showed a mean drop in visual acuity and recommended that we should evaluate the need for grid laser in select cases.

- **Ophthalmology. 1997 Sep;104(9):1433-41.**

Diode laser (810 nm) versus argon green (514 nm) modified grid photocoagulation for diffuse diabetic macular edema

Olk RJ et al compared argon green against diode laser for treatment of CSME in 171 eyes with a mean follow up of twelve months. They reported no statistically significant difference in any of the parameters between the two groups. The various parameters used to deteremine the

efficacy were improvement in vision, drop in vision, reduction in macular edema and the number of retreatments required. However they noted an important finding that eyes with cystoids macular edema had a poorer outcome than patients who did not have any cystoids macular edema. The also noted that an associated systemic vascular disease can have an adverse effect on the outcome of treatment with either laser.

- **Ophthalmology. 1990 Sep;97(9):1101-12;**

Argon green (514 nm) versus krypton red (647 nm) modified grid laser photocoagulation for diffuse diabetic macular edema.

Olk RJ studied two hundred and twenty five eyes of CSME treated by the above mentioned lasers. In a 2 year follow up period they found no statistically significant difference among the two lasers. They assessed various parameters like visual acuity, reduction in macular edema, number of retreatments required and changes in visual field. In all the above mentioned parameters there was no difference between the two lasers emphasising the fact that both have a similar effect in the treatment if diabetic macular edema.

AIMS AND OBJECTIVE

To determine whether the 577 nm yellow laser can provide a better treatment outcome when compared with the conventional 532 nm green laser for diabetic macular edema.

Intervention(s) in this Clinical Trial

- Device: 577 nm yellow laser

Laser to be administered at the time of enrollement in the study and may be repeated at 16 weeks if there is treatment failure.

- Device: 532 nm green Frequency doubled Nd:Yag laser

Laser to be administered at the time of enrollement in the study and may be repeated at 16 weeks if there is treatment failure.

Groups of the Trial

- Experimental: 577 nm yellow laser

- Active Comparator: 532 nm green Frequency doubled Nd:Yag laser

Outcome Measures for this Clinical Trial

• Best corrected visual acuity by logarithm of the minimum angle of resolution (LogMAR) compared to pre laser vision and vision at 4 months post laser

•Macular thickness measured by optical coherence tomography (OCT) comparing pre laser macular thickness and thickness measured by OCT at 4 months post laser

MATERIALS AND METHODS

A sample of 71 eyes from 54 patients, 36 males and 18 female patients between 44 to 76 years of age, who fit the criteria for the study were included in the study during the recruitment period between January 2011 to December 2011 and the follow up period extended upto June 2012. The total study duration was of 18 months.

INCLUSION CRITERIA:

- Age >= 18 years.
- ⦿ Diagnosis of diabetes mellitus (type 1 or type 2) or a documented diabetic who is currently using insulin or oral hypoglycemic agents
- ⦿ DIAGNOSIS OF CSME as documented clinically by a experienced retinal surgeon along with proof of its presence in OCT.

EXCLUSION CRITERIA:

- ⦿ Patient having significant renal disease i.e. a case of chronic kidney disease that mandates regular dialysis or kidney transplant.
- ⦿ An uncontrolled systemic hypertensive with a systemic Blood pressure > 180/110.

- The following exclusions apply to the study eye only (i.e., they may be present for the non study eye):
- Vitreo macular traction as diagnosed by OCT or clinically
- Concurrent proliferative diabetic retinopathy.
- Associated ocular morbidity that would preclude restoration of visual acuity even after resolution of macular edema (e.g., foveal atrophy, subfoveal hard exudates, pigment abnormalities, significant macular ischemia caused be vein occlusions or diabetes, optic atrophy or other optic nerve head disorders).
- A coexistent ocular condition is present (other than diabetic retinopathy) that by its natural course might **affect macular edema** or alter visual acuity during the course of the study (e.g., vein occlusion, uveitis or any ocular inflammatory disease, epiretinal membrane, neovascular glaucoma, etc.).
- **Substantial cataract** that is likely to be decrease visual acuity by 3 lines or more
- History of **focal or grid macular photocoagulation** within six months prior to enrolment.

- History of use of periocular or intraviteral steroids within six months prior to enrolment.
- History of having been administered intravitreal ANTI VEGF agents within six months prior to enrolment.
- History of panretinal (scatter) photocoagulation (PRP) prior to enrollment.
- History of major ocular surgery within prior 6 months or anticipated within the next 6 months.
- Patients who will be unable to come for a follow up visit at 4 months post laser.

Sample Size Calculation: As there was no previous published reports on the comparative efficacy of 532nm frequency doubled Nd:Yag green laser versus the 577nm solid state yellow laser, comparative analysis of other lasers were sought and sample size was brought about by the number of cases which fit the inclusion and exclusion criteria within the recruitment period of one year from January 2011 to December 2011.

METHODOLOGY

A data sheet was attached to the case sheets of the patients enrolled in the study. The data sheet contains a detailed patient history regarding the duration of defective vision, the duration of diabetes mellitus, history of prior ocular disease, history of any surgery or procedure performed in the study eye.

The visual acuity at enrolment was recorded using a Snellen's visual acuity chart. The Snellen's visual acuity was then converted to logarithm of the minimum angle of resolution (LogMAR) scale at the time of statistical analysis.

Patient then undergoes a detailed slit lamp examination and the anterior segment findings including lens status was recorded.

A thorough fundus examination was done by a retina specialist and the diabetic Retinopathy was graded according to the ETDRS Classification of diabetic retinopathy and the presence of clinically significant macular edema was recorded.

Basic blood investigations including postprandial blood sugar, Hemoglobin A1C, Blood Urea And Serum Creatinine was done for all patients.

A fundus photograph of the patient was taken and a Fundus fluoroscein angiography was done after obtaining a physician s fitness.

A Ocular coherence tomography of the study eye was performed using Zeiss Cirrus SD-OCT and the foveal and/or para foveal thickness depending on the area and extent of involvement was noted.

The patient was then randomized by a table of random numbers and assigned to either the experimental group (577nm yellow laser) or into the active comparator group (532nm green laser).

Informed consent was obtained from the patient. Pupils were fully dilated using 1% tropicamide eye drops. Topical anaesthesia was attained by using 2% xylocaine, 3-4 application before the procedure. Volk contact lenses were used for magnifications, controlling the globe and laser delivery. The 532 nm green laser was delivered using the Iridex laser delivery system. The 577nm yellow laser was administered using the Iridex laser delivery system. The laser power was titrated according to the extent of thickness of the macular edema. The number of spots was also varied between the patients according to the area of involvement of the macula. The duration of the laser spot delivary was fixed at 0.1 second. The laser was administered either as a focal laser for focal macular edema or as a modified grid laser for diffuse macular edema. The

modified grid pattern was given as a C pattern with laser burns given in 2 -3 rows superior, temporal and inferior to the fovea with a minimum of 500 microns distance away from the centre of the fovea sparing the papillomacular bundle.

The patients were then asked to review at 4 months post laser. At follow up the visual acuity was recorded with Snellen's visual acuity chart. A detailed slit lamp and 90 D fundus examination was done and the macular thickness (foveal or parafoveal) using the Carl Zeiss Spectral Domain OCT was recorded. In case of worsening of macular thickness a repeat laser of the same type as the one administered in the first visit was considered at 4 months post laser to improve the treatment outcome.

RESULTS AND ANALYSIS

A prospective randomized study was done on 71 eyes of 54 patients whose visual acuity and macular thickness by OCT was recorded prior to and 4 months after application of laser, conducted at the Vitreo Retina Clinic, Aravind Eye Hospital, Madurai between Jan 2011 to June 2012.

Statistical analysis

The Statistical analysis was performed using STATA 11(Stata Corp TX College Station USA).

The continuous variables are described as Mean ± Standard deviation, and the categorical variables are described as frequency and percentage.

Student Independent sample t-test was used for age, HbA1C and power for find the statistical significance between green and yellow laser.

Wilcoxon sign rank was used for Log MAR Visual acuity, Foveal and parafoveal measurements of Baseline and follow up (four months) were compared and used the students paired t-test. $P<0.05$ is considered as statistically significant

1. **Age:**

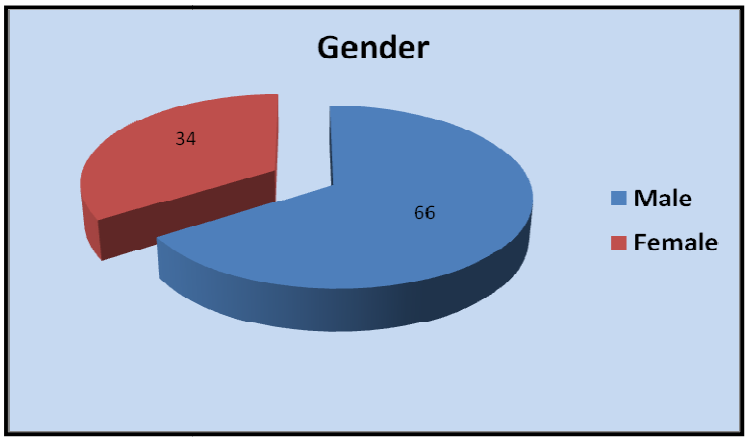
The age of the patients in the two arms of the study ranged between 44 to 76 years.

The mean age of patients in the Green laser group was 56.94 years.

The mean age of patients in the yellow laser group was 56.13 years.

2. **Sex Ratio:**

	Frequency	Percentage
Male	36	66
Female	18	34
Total	54	100

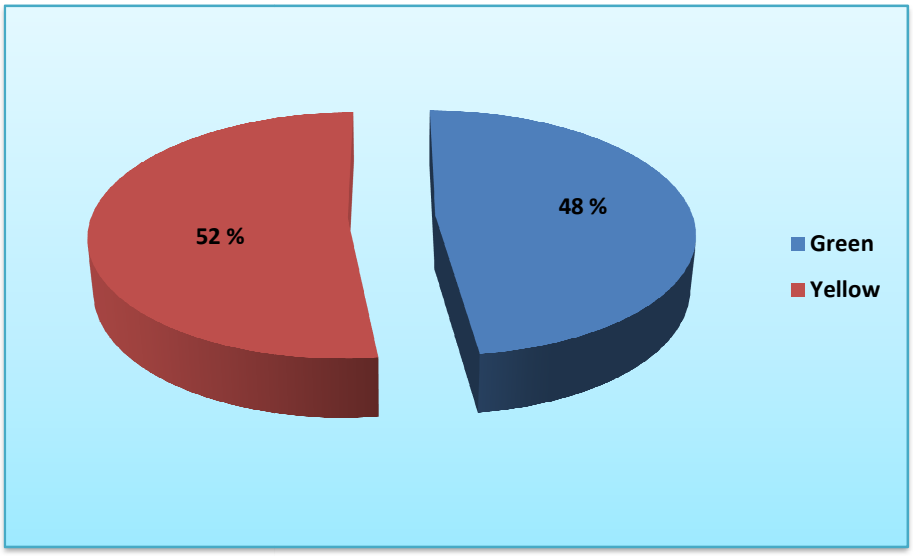


There were 36 males(66%) and 18 female patients(34%) distributed among the two arms of the study.

3. Number of eyes in each group:

The eyes of individual patients were randomized to each group by a table of random numbers and there were 34 eyes (48%) in the green laser group and 37 eyes (52%) in the yellow laser group.

	Frequency	Percentage
Green	34	48
Yellow	37	52
Total	71	100



4. **Hemoglobin A1C:**

Hemoglobin A1c was measured in all 54 patients in the two groups.

The mean measured HbA1c in the green laser group was 6.81 with a standard deviation of 0.99.

The mean measured HbA1c in the yellow laser group was 6.70 with a standard deviation of 1.56.

5. **Diabetic Retiopathy Status:**

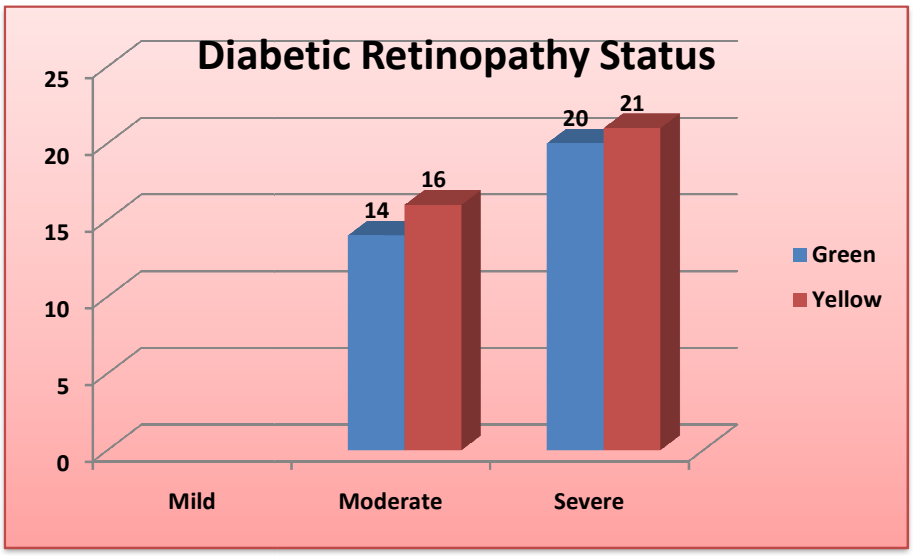
All 54 patients either had moderate or severe NPDR.

30 of the 71 eyes had moderate NPDR with 14 eyes in the green laser group and 16 eyes in the yellow laser group.

The remaining 41 eyes of the 71 eyes had Severe NPDR with 20 eyes in the green laser group and 21 eyes in the yellow laser group.

Diabetic retinopathy status:

	Green	Yellow	Total
Mild	--	--	--
Moderate	14	16	30
Severe	20	21	41
Total	34	37	71



6. Visual Acuity:

The visual acuity was recorded by Snellen s Visual Acuity chart at a distance of 6 metres, at baseline, prior to application of laser and was recorded again at 4 months review. The Visual acuity was then converted to the Standard LogMar scale prior to statistical analysis.

Visual Acuity

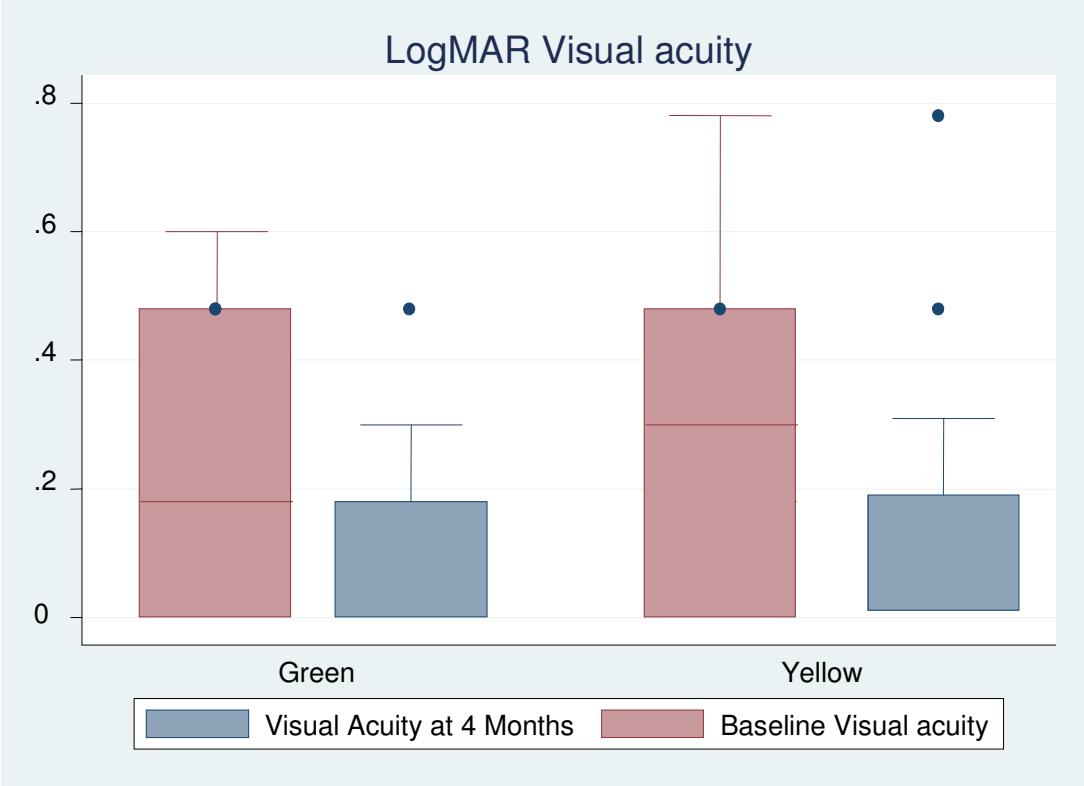
	Baseline	4 Months (After Laser)	P-Value
	Median (IQR)	Median (IQR)	
Green	0.18 (0.48)	0.18(0.18)	0.002
Yellow	0.3(0.48)	0.18(0.18)	<0.001

The Median Visual acuity at baseline in the Green laser group was 0.18(0.48) and 0.3(0.48) in the yellow laser group as measured by LogMar scale.

At 4 months after laser the median visual acuity in the green laser group had improved to 0.18 which was statistically significant with a p value of 0.002.

At 4 months after laser the median visual acuity in the yellow laser group had improved to 0.18 which was statistically significant with a p value of <0.001.

There was however no statistically significant difference between the degree of improvement of visual acuity between green and yellow laser. Thus the green and yellow laser had no difference in the efficacy of improvement of visual acuity post laser.



There are 3 outliers in the above graph. They are the 3 patients who required repeat laser at 4 months post laser due to unsatisfactory improvement or deterioration in any one of the outcome variables namely visual acuity and measured macular thickness on OCT. 2 of these patients belonged to the green laser group and 1 patient from the yellow laser group required repeat laser at 4 months post laser. All three patients showed significant improvement in the macular thickness as measured by OCT at 4 months post repeat laser.

7. Power setting used in each laser system:

The mean power of laser used in the green laser group was 112mW with a standard deviation of 18mW.

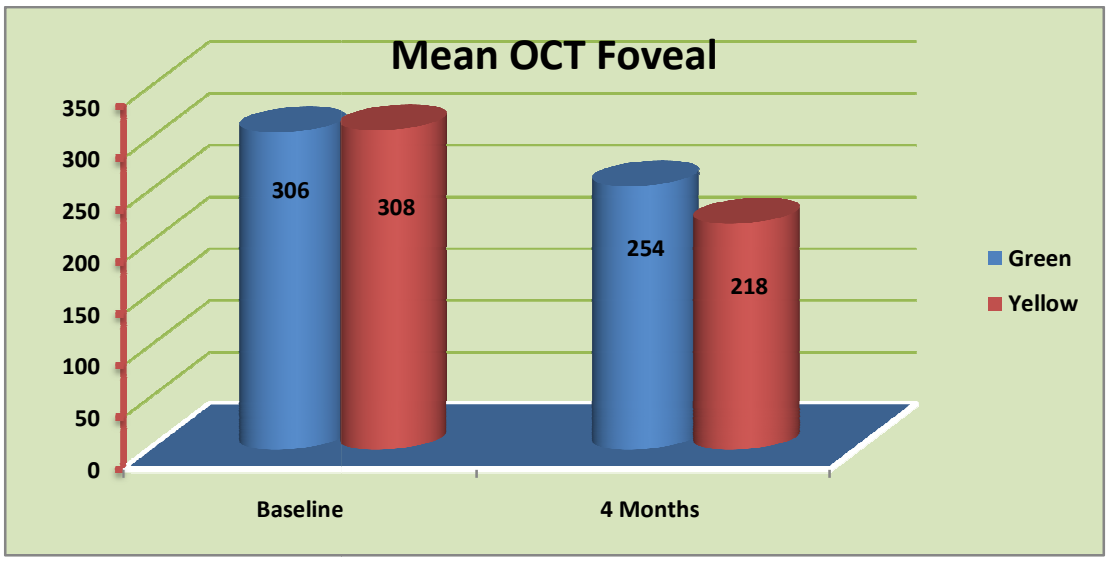
The mean power of laser used in the yellow laser group was 111mW with a standard deviation of 21 mW.

8. Optical Coherence Tomography Results:

8a. Foveal:

	Baseline	4 Months (After Laser)	P-Value
	Mean ± SD	Mean ± SD	
Green	306.21 ± 133	254.44 ± 105.27	<0.001
Yellow	308 ± 131	218 ± 66	<0.001

The mean measured foveal thickness by OCT in the green laser group was 306.21 microns with a standard deviation of 133 microns. At 4 months post laser the foveal thickness had decreased to 254.44 microns with a standard deviation of 105.27 microns. This showed a statistical significant difference in the improvement in the macular thickness at 4 months post green laser with a p value of <0.001.

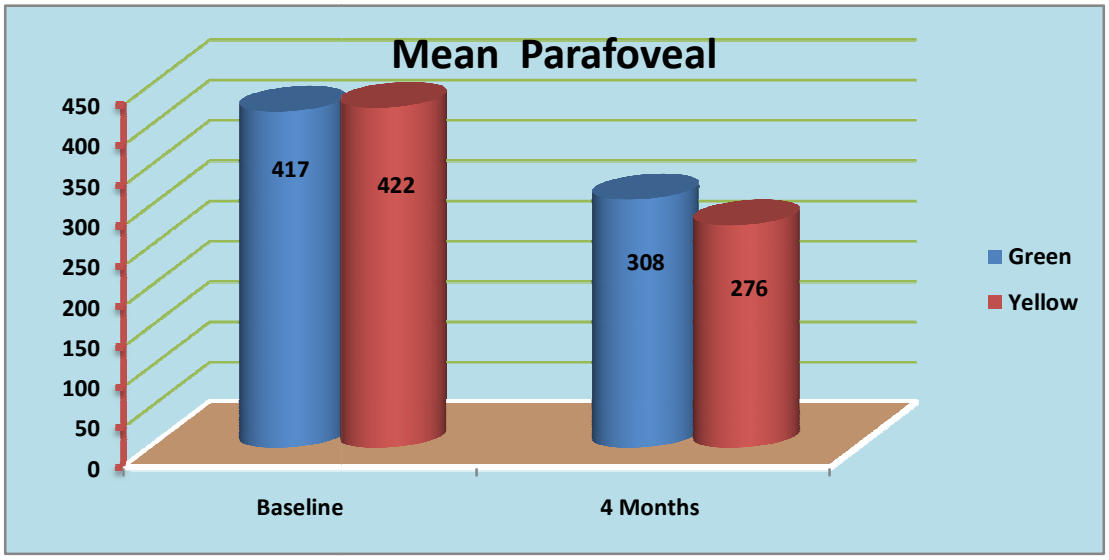


The mean measured foveal thickness by OCT in the yellow laser group was 308 microns with a standard deviation of 131 microns. At 4 months post laser the foveal thickness had decreased to 218 microns with a standard deviation of 66 microns. This showed a statistical significant difference in the improvement in the macular thickness at 4 months post yellow laser with a p value of <0.001.

8.b Parafoveal thickness comparison between pre and post laser using OCT

	Baseline	4 Months (After Laser)	P-Value
	Mean ± SD	Mean ± SD	
Green	417 ± 75	308 ± 72	<0.001
Yellow	422.35 ± 65	276 ± 41	<0.001

The mean measured parafoveal thickness by OCT in the green laser group was 417 microns with a standard deviation of 75 microns. At 4 months post laser the parafoveal thickness had decreased to 308 microns with a standard deviation of 72 microns. This showed a statistical significant difference in the improvement in the parafoveal macular thickness at 4 months post green laser with a p value of <0.001.



The mean measured parafoveal thickness by OCT in the yellow laser group was 422.35 microns with a standard deviation of 65 microns. At 4 months post laser the parafoveal thickness had decreased to 276 microns with a standard deviation of 41 microns. This showed a statistical significant difference in the improvement in the macular thickness at 4 months post yellow laser with a p value of <0.001.

9. Comparison of measure of reduction in foveal thickness after using green and yellow laser:

9A. Foveal thickness

	Green Laser	Yellow	
Foveal			
% of Reduction	17%	29%	0.135

The green laser caused a 17% reduction in foveal thickness over a period of 4 months. In the same time interval yellow laser showed a slightly better efficacy with an improvement in foveal thickness by 29%. But the difference in the reduction of foveal thickness between the two laser was not statistically significant with a p value of 0.135.

9B. Parafoveal thickness

	Green	Yellow	P-Value
Parafoveal			
% of Reduction	26%	35%	0.411

The green laser caused a 26% reduction in parafoveal thickness over a period of 4 months. In the same time interval yellow laser showed a better efficacy with an improvement in parafoveal thickness by 35%. But the difference in the reduction of parafoveal thickness between the two laser was not statistically significant with a p value of 0.411.

DISCUSSION

ETDRS firmly established the role of laser in the treatment of diabetic macular edema [173]. Over the years several lasers have been developed each of which have shown different levels of efficacy in treating diabetic macular edema. Despite the development of newer agents like intra vitreal triamcinolone and Anti VEGF drugs, laser continues to remain the definitive and gold standard treatment of diabetic macular edema.

Our Study included a total of 71 eyes of 54 patients who fit the inclusion criteria between the period January to December 2011. This is comparable to other studies where they have compared the efficacy of two laser groups in treating diabetic macular edema. A study by Fugueira et al [181] comparing the efficacy of micropulse diode laser with conventional green had 84 eyes from 53 patients. Another study by Karacorlu et al(2) comparing the efficacy of argon green with dye yellow laser had a total number of 85 eyes.

The age range of the patients included in our study was from 44 to 76 years with the mean age in the green laser group being 56.94 years and that in the yellow group was 56.13 years with a very similar age distribution of patients in the two groups. All the patients in our study were Type II diabetics. A study by JK Lutrull et al [182] had an age range of

42 to 90 years with the mean age being 56 years. Our study comprised of 36 males and 18 females with a sex ratio of 2:1.

It has been well established that HbA1C has a definitive diagnostic role to play in the control of diabetes of mellitus and patients with very high values of HbA1C are at risk of progression of Diabetic Retinopathy despite adequate laser treatment. So a cut off value of HbA1C of 10% was taken in our study. This value was similar to the criteria taken in previous comparative studies by Laursen et al [180] and Sivaprasad S et al [182]. In our study we had patients with fairly controlled diabetes with the mean HbA1C in the green group being 6.81% and that in the yellow group being 6.70%.

The patients were randomized in the two groups and after consent for laser was taken underwent laser as assigned in the two groups.

The exact mechanism of grid laser photocoagulation is unknown. Electron microscopic studies in monkeys and rabbits were the basis for suggesting that the mechanism involved in photocoagulation might be an opening up of new pathways by disrupting the retinal pigment epithelial diffusion barrier, thus allowing the egress of fluid from the retina or subretinal spaces across the retinal pigment epithelium as evidenced by

the passage of tracer material from the choriocapillaries into the subretinal space. [174,175,176]

Others suggested that the mechanism of grid photocoagulation might be "photocoagulation debridement of disordered retinal pigment epithelium" ultimately leading to removal of the disordered or sick retinal pigment epithelial cells and replacement by a young more vigorous population of cells with better RPE pumping activity [177]. Another possible explanation is that the laser may directly act on the leaking retinal microaneurysms causing their closure by photocoagulation and preventing further leakage. The lasers are absorbed by the melanin and xanthophylls pigments in the retinal pigment epithelium for their action to occur [179]. Both green and yellow laser have low absorption by melanin pigments(1). The distinct advantage of yellow laser is that it has almost no affinity towards macular xanthophylls pigments and thus causes minimal phototoxicity. It also has a high affinity for oxy haemoglobin(1) which helps in focal laser as the yellow wavelength is absorbed by the oxy haemoglobin in the retinal microaneurysms leading to their closure.

The baseline visual acuity across the two groups as measured by LogMar Visual Acuity scale was 0.18 in the green group and 0.3 in the yellow group. At 4 months post laser the visual acuity was stable or better in 68 of the 71 eyes. Only 3 eyes had a decrease in visual acuity necessitating

repeat laser treartment. Thus 94.4% of patients had a stable or better visual acuity than at baseline. The patients from both laser groups had a statistically significant improvement in vision post laser treatment. The results produced in terms of stable vision and re treatments required were similar to the study by JK Lutrull et al(182) where they have shown statistically significant improvement in vision. In the study by Karacorlu et al [2] there was a drop in vision in 12 of 85 eyes after using either argon green or dye yellow laser.

No statistically significant difference was noted in the outcome of visual acuity between green and yellow laser. Other reports too by Karacorlu et al [2] showed no additional benefit of dye yellow laser over conventional green laser in relation to visual outcome. A study by Fuguiera et al[181] also showed no significant difference in the outcome of visual acuity when two laser of different wavelengths were used to treat diabetic macular edema.

The power of the laser setting to cause laser photocoagulation of mild intensity in our study was 112mW in the green laser group with a standard deviation of 18mW and in the yellow laser group was 111mW with a standard deviation of 21 mW. There was no statistically difference between the two groups in the amount of power required to treat diabetic macular edema. A study by Karacorlu et al [2] says that they required 20%

less power in the dye yellow laser group as compared to the argon green laser group during grid photocoagulation.

On measurement of macular thickness on Optical Coherence Tomography showed a statistically significant decrease in the amount of macular edema both foveal and perifoveal after treatment with laser in both the treatment groups.

The green laser caused a 17% reduction in foveal thickness over a period of 4 months. In the same time interval yellow laser showed a slightly better efficacy with an improvement in foveal thickness by 29%. But the difference in the reduction of foveal thickness between the two laser was not statistically significant with a p value of 0.135. The perifoveal thickness also reduced significantly with a reduction of 26% in the green laser group and further reduction of 35% in the yellow laser group. Though both lasers showed very significant decrease in perifoveal thickness with a slightly better improvement in the yellow laser group, this difference was not statistically significant.

No untoward complications like loss of central vision, choroidal neovascularisation, subretinal fibrosis, exudates migration to the fovea was found during the duration of the study and follow up.

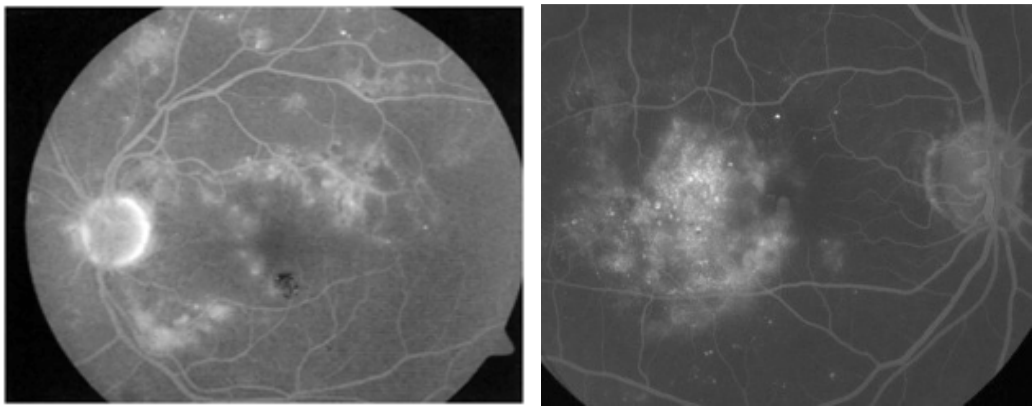
LIMITATIONS OF THE STUDY

1. Though yellow laser showed some advantage over the green laser in reducing the quantum of macular edema on OCT, the size of the study was not enough to produce a statistically significant difference.
2. The use of more objective measures like microperimetry which is not available at our institute would help in detecting the extent of macular scotomata caused by the laser and help in assessing the quantity of improvement in vision.
3. Other measures of vision like contrast sensitivity and quantitative measures of colour vision like Fransworth Munsell 100 hue test could bring out subtle differences.
4. A longer follow up period is required to test the stability of visual improvement produced by the yellow laser and also to look for long term complications.
5. This study did not include patients who had been administered intravitreal triamcinolone and intravitreal Anti VEGF drugs. A more comprehensive study to compare the laser uptake and efficacy following this intravitreal agents is called for.
6. This study also did not compare the outcome of individual lasers in treating spongiform against cystic edema as shown by OCT.

CONCLUSION

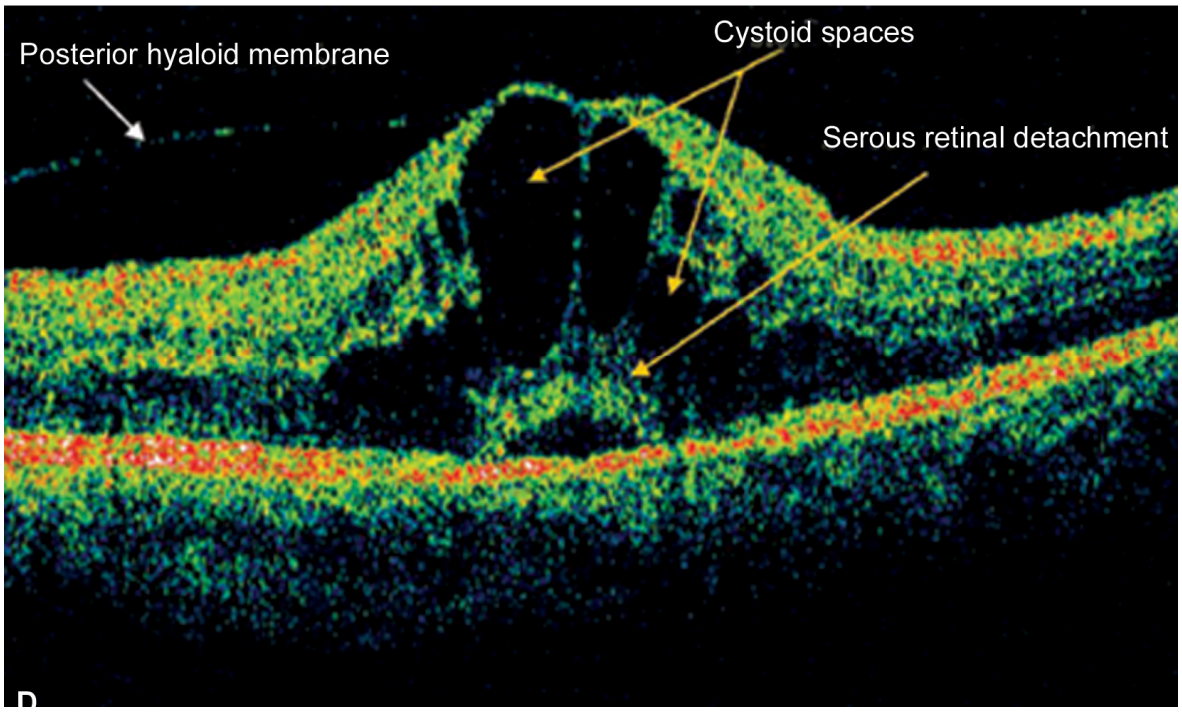
The study emphatically reiterates the role of laser alone in the treatment of diabetic macular edema in carefully chosen cases. In well controlled diabetics laser continues to remain the gold standard in the treatment of diabetic maculopathy. No statistically significant advantage was shown for the 577nm solid state yellow laser over the 532nm frequency doubled Nd:Yag laser in reducing macular edema but a positive trend was shown which needs a larger study group and a more prolonged follow up to detect accurate and more minute differences.

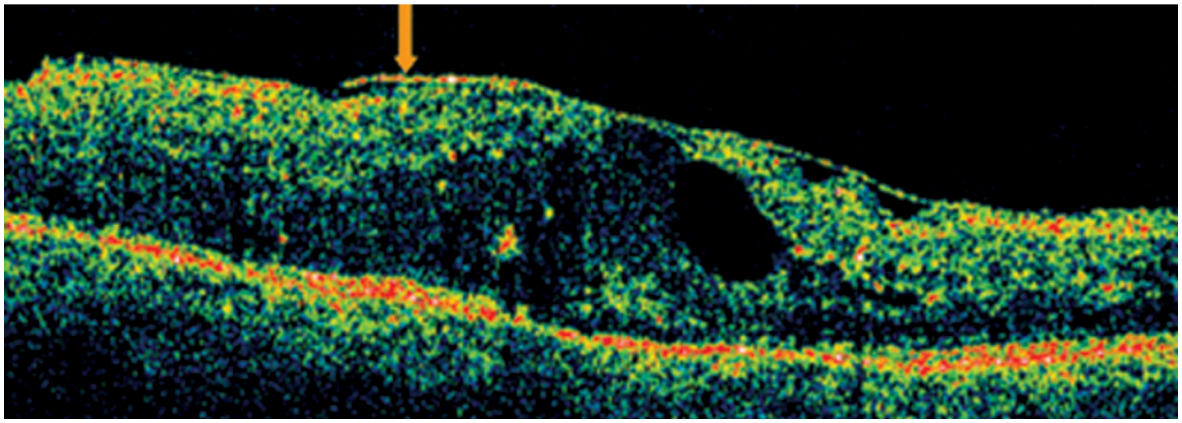
FUNDUS PHOTO SHOWING CSME



FFA SHOWING FOCAL AND DIFFUSE LEAKS

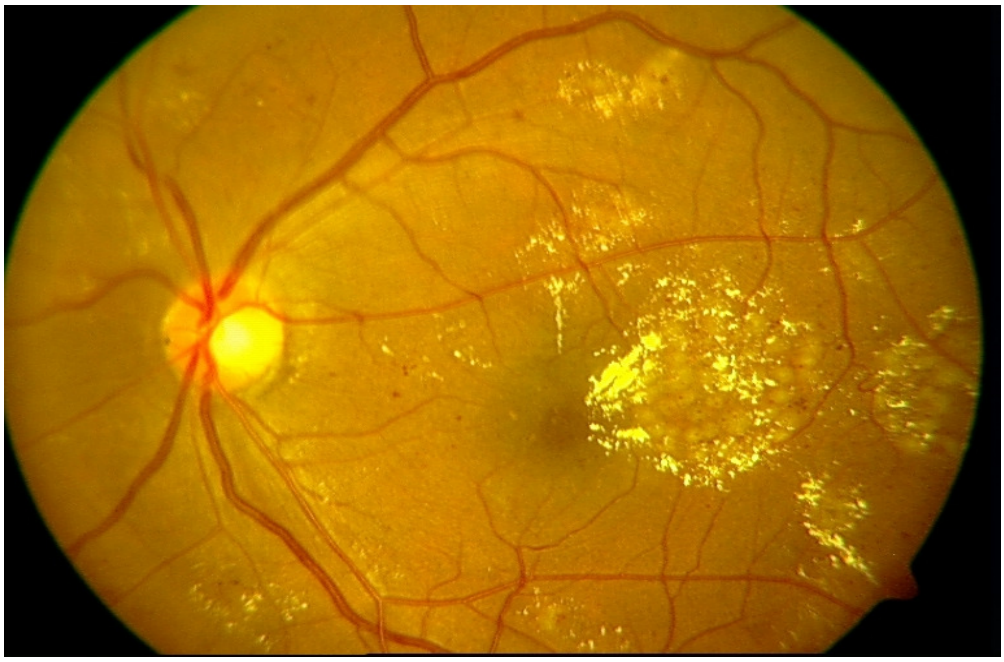
OPTICAL COHERENCE TOMOGRAPHY

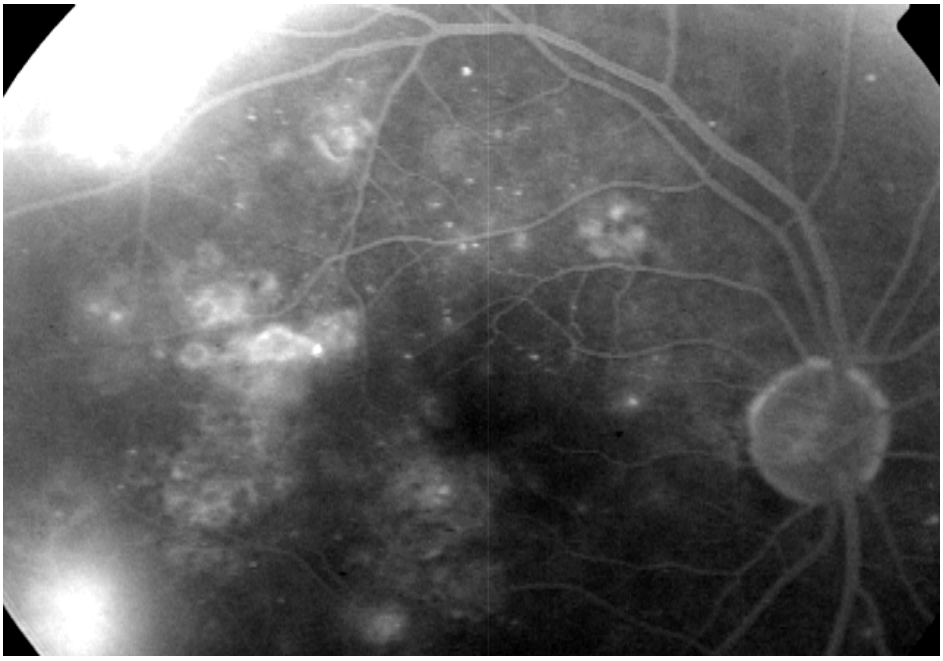




OCT SHOWING SPONGIFORM EDEMA

CSME – POST FOCAL LASER TREATMENT





COMPARISON OF COMMONLY USED LASER WAVELENGTHS				
CHARACTERISTICS*	ARGON BULE – GREEN (488 nm)	ARGON GREEN (514 nm)	DYE YELLOW (577 nm)	KRYPTON RED (647 nm)
Hemoglobulin absorption	80%	78%	98%	5%
Macular xanthophylls Absorption #	59%	11%	0%	0%
Melanin (RPE) # absoption	50%	45%	40%	35%
Transmission through ocular media Δ	29%	35%	47%	50%

***All percentages given are approximations**

 # Depends on light versus dark fundi.

Δ Depends on age.

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PROFORMA

- NAME: STUDY NO:
  - GROUP:
- AGE:
- SEX:
- ADDRESS:

- MR. NO:
- TELE NO:

HISTORY

- DEFECTIVE VISION: _____DAYS
- EYE:

R	L

- DURATION OF DM:_____YEARS
- TYPE OF DM

I	II

RELEVANT OCULAR HISTORY:

H/0 ANY OCULAR PROCEDURE/SURGERY DONE BEFORE TO THE STUDY EYE(s):

SYSTEMIC DISEASE: cardiac, HT, asthmatic, renal problem, allergy, others

BLOOD INVESTIGATION(s):

FBS/PPBS:

HBA1c:

LIPID PROFILE:

BLOOD UREA:

SERUM CREATININE:

VISUAL ACUITY (BCVA)

VISUAL ACUITY	RIGHT	LEFT
PRE LASER		
4^{TH} MONTH		
6^{TH} MONTH		

- SLIT LAMP FINDINGS:

R	L

- FUNDUS DIAGNOSIS: stage of DR

RIGHT EYE	LEFT EYE

- CONSULTANT:_____
- FUNDUS PHOTO:

- FFA :

- OCT – MACULAR THICKNESS

1^{ST} DAY		4^{TH} MONTH	
RE	LE	RE	LE

- LASER DETAILS 1ST DAY 2ND

DATE		
PROCEDURE		
POWER		
DURATION		
SPOT SIZE		
NO OF SPOTS		

COMPLICATIONS IF ANY NOTED DURING ANY OF THE FOLLOW UPS:-

VISUAL ACUITY & OCT – MACULAR THICKNESS AT FINAL FOLLOWUP

VISUAL	ACUITY	OCT	MACULAR THICNESS
RE	LE	RE	LE

MATER CHART

sno	group	name	age	sex	address	mrno	hba1c	study_eye	bcva	bcva_review	anterior_seg	fundus_diagnosis	baseoct_fovea	baseparafov	basepower	duration_ms	base_spots	va_4m	va_4mr	octfovea_4m	octpara_4m	parameters - repeat laser	Final outcome
1	Yellow	Velayudhan	58	M	Ezhachery, Kottayam	3087774	6.5	RE	R-6/9	6/9	early imc	R:Mod NPDR/CSME	496		100	100	25	RE 6/9	6/9	284			
2	Green	Savithri	55	F	Mattaparai, Dindigul	3098148	5.8	RE	R-6/18	6/18	early IMC	RE: Mod NPDR/CSME	243	507	90	100	55	RE 6/9	6/9	210	380		
3	Green	Savithri	55	F	Mattaparai, Dindigul	3098148	5.8	LE	LE - 6/9p	6/9	Early IMC	LE: Mod NPDR/CSME	235	455	90	100	32	LE 6/9	6/9	216	340		
4	Yellow	Dhanapackiam	52	F	Kambilampatti, Dindigul	3098234	6.8	RE	R-6/12P	6/12	lens changes	RE: Mod-Severe NPDR/CSME Hard Exudat	290		110	100	111	RE 6/9	6/9	171			
5	Green	Dhanapackiam	52	F	Kambilampatti, Dindigul	3098234	6.8	LE	L-6/6P	6/6	lens changes	LE: Mod NPDR/CSME	452		100	100	81	LE 6/6p	6/6	239			
6	Green	Shahul Hameed	62	M	Athirampatinam, Tanjore	3065765	7.2	RE	R - 6/18	6/18	IMC (PSCC)	RE- severe NPDR/CSME	370		100	100	39	RE 6/12	6/12	254			
7	Green	Manickam	70	M	Athoor, Dindigul	3078366	4.9	LE	le - 6/6p	6/6	wnl	L: ModNPDR/CSME	484		110	100	25	LE 6/6	6/6	420			
8	Yellow	Ramamoorthy MS	42	M	Aruuppukottai, Virudunagar	3100503	5.5	RE	RE 6/24	6/24	wnl	RE: severe npdr/csme		469	90	100	62	re 6/9	6/9	310			
9	Green	Ramamoorthy MS	42	M	Aruuppukottai, Virudunagar	3100503	5.5	LE	LE 6/24p	6/24	WNL	LE: severe npdr/csme		647	70	100	21	re 6/12	6/12	416			
10	Yellow	Salahudeen	60	M	Kollam, Kerela	3109224	6.4	RE	R 6/12p	6/12	wnl	Re: Mod NPDR CSME	423		120	100	38	RE 6/6p	6/6	226			
11	Green	Dharmarajan	65	M	Santhaipettai, Madurai	3168421	6.9	RE	Re: 6/24	6/24	lens changes	Re: Severe NPDR CSME with CME PVD	660		120	100	42	RE 6/18	6/18	480			
12	Yellow	Maragatham	40	F	Nethaji Road, Madurai	3106807	7.2	RE	Re 6/9p	6/9	Wnl	BE: Mod NPDR/CSME	188	399	160	100	11	RE 6/9	6/9	186	348		
13	Yellow	Suganthan	61	M	Varkala, Trivandrum	3133977	8.3	LE	LE 6/12	6/12	LC	LE: SEVERE NPDR/CSME	334		90	100	25	RE 6/9p	6/9	214			
14	Yellow	Subramnaniam	63	M	jayanthi Nagar, Madurai	3156654	7.9	RE	re 6/12p	6/12	wnl	BE: Mod NPDR/CSME	311	511	150	150	47	RE 6/9	6/9	220	344		
15	Yellow	Aysha Beevi	56	F	Thasiladar Nagar, Madurai	3167633	8.4	LE	LE 6/18	6/18	wnl	BE: Mod-Severe NPDR/CSME	309	482	120	100	24	LE 6/12	6/12	190	312		
16	Yellow	Premchand	50	M	Trivandrum	3190307	8.6	RE	re 6/9	6/9	wnl	BE: Mod-Severe NPDR/CSME	346		120	100	42	RE 6/9	6/9	391		110, 100, 51	270u
17	Yellow	Rabiyammul	72	F	Vilachery, Madurai	3193007	5.2	RE	re 6/24p	6/24	ns II	BE: Mod-Severe NPDR/CSME	296	400	140	120	41	RE 6/9	6/9	170	320		
18	Green	Kunhaysha	70	F	Vadakara, Kozhikode	3204785	6.4	LE	le 6/12	6/12	lc	BE: Severe NPDR/CSME	207	223	150	100	49	LE 6/9	6/9	180	112		
19	yellow	Indira	56	F	Kandalloor, Allepey	3208489	8.9	RE	re: 6/36	6/36	lc	BE: Severe NPDR/CSME	630		100	100	60	RE 6/18	6/18	415			
20	yellow	Indira	56	F	Kandalloor, Allepey	3208489	8.9	LE	le: 6/6p	6/6	lc	BE: Severe NPDR/CSME	188	328	100	100	34	LE 6/6	6/6	160	265		
21	yellow	Seshan	52	M	Simmakkal, Madurai	2906835	8.1	RE	re: 6/36p	6/36	early imc	BE: Severe NPDR/CSME	603		120	100	102	RE 6/36	6/36	384			
22	Green	Kalaiselvi	48	F	Annamalai Nagar, Tanjore	3143412	6.6	RE	re 6/18	6/18	imc	be: severe npdr/csme	319		100	100	29	RE 6/9	6/9	250			
23	Green	Kalaiselvi	48	F	Annamalai Nagar, Tanjore	3143412	6.6	LE	le 6/24	6/24	imc	be: severe npdr/csme	375		120	100	42	LE 6/18p	6/18	363			
24	Green	Seshan	52	M	Simmakkal, Madurai	2906835	8.1	LE	le: 6/12p	6/12	early imc	BE: Severe NPDR/CSME	415		130	100	94	LE 6/12	6/12	319			
25	Yellow	Abdul Wahab	65	M	Mudukulathur, Ramanathapuram	3128738	6.2	LE	le: 6/9	6/9	wnl	Be: Mod NPDR/CSME	248	453	140	100	46	LE 6/9p	6/9	186	290		
26	Yellow	Anthony P.C.	74	M	Ramakkal Medu P.O., Idukki, Kere	3214535	5.5	RE	re: 6/12p	6/12	early imc	Be:Severe NPDR/CSME	363		150	100	61	RE 6/12	6/12	250			
27	green	Karishma Begum	55	F	Sattankulam, Ramanathapuram	3184299	5.0	LE	le 6/12	6/12	lc	LE: Mod npdr csme	255	439	110	100	40	LE 6/9	6/9	210	294		
28	green	Kesari das	48	F	Thiruvarambu, Kanyakumari	3017220	8.0	RE	6/6p	6/6	lc	BE: Mod-Severe NPDR/CSME	241	391	140	100	36	le 6/6	6/6	160	265		
29	green	Ayyasamy	73	M	Valangaiman, Thiruvarur	3204341	8.6	RE	re: 6/18p	6/18	pciol	BE: Severe NPDR/CSME	625		110	100	34	re 6/9p	6/9	600		100, 100, 41	390u
30	Yellow	Paneer Selvam	58	M	Virudhunagar	3235638	6.4	RE	re 6/18	6/18	wnl	BE: Severe NPDR/CSME	410		90	100	42	Re 6/9p	6/9	240			
31	Yellow	Durairaj	42	M	Ramanathapuram	3128742	5.8	RE	re 6/6p	6/6	wnl	Re: Mod Npder with CSME	640		100	100	28	RE 6/6	6/6	218			
32	Yellow	Anthony P.C.	74	M	Ramakkal Medu P.O., Idukki, Kere	3214535	5.5	LE	le: 6/18	6/18	early imc	BE: Severe NPDR/CSME	420		150	100	52	LE 6/12	6/12	242			
33	Yellow	Chandra Devi	50	F	Mettupatti, Vadipatti	2218268	6.9	RE	re: 6/6p	6/6	wnl	Be: Mod NPDR/CSME	170	327	130	100	84	RE 6/6	6/6	168	244		
34	green	Lakshmanan	40	M	Thiruppalai, Madurai City	3235646	6.4	RE	re: 6/6	6/6	wnl	Be: Mod NPDR/CSME	199	383	100	100	53	RE 6/6	6/6	180	242		
35	Yellow	Chandra Devi	50	F	Mettupatti, Vadipatti	2218268	6.9	LE	le: 6/6p	6/6	wnl	Be: Mod NPDR/CSME	327	431	130	100	72	RE 6/6	6/6	217	311		
36	green	Ayyanar	52	M	Vellipattinam, Ramanathapuram	3240581	6.9	LE	le: 6/9	6/9	lens changes	LE: Mod npdr csme	184	440	120	100	78	le 6/6p	6/6	182	360		
37	green	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	7.1	RE	re:6/9	6/9	lc	re: Mod - Severe NPDR/CSME	327	519	120	100	48	Re 6/6	6/6	240	410		
38	yellow	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	7.1	RE	le:6/9p	6/9	lc	le: Mod - Severe NPDR/CSME	237	455	100	100	62	Re 6/6	6/6	186	270		
39	Yellow	Abdul Azeez	43	M	Malapuram, Kerala	3235639	7.4	RE	re: 6/6p	6/6	WNL	BE: Severe NPDR/CSME	245	480	120	100	51	Re 6/6	6/6	211	326		
40	Yellow	Ponnammal	66	F	Aruppukottai, Virudhunagar	3174419	5.6	RE	re: 6/12p	6/12	pciol	re: Mod - Severe NPDR/CSME	141	430	100	100	135	Re 6/9	6/9	136	260		
41	Yellow	Jose K.J	59	M	Kottayam	3253919	7.2	RE	re: 6/24	6/24	early pscc	re: Disc pallor/ Severe NPDR/CSME	163	435	80	100	80	RE 6/18	6/18	156	230		
42	Yellow	Ramalingam	63	M	Samiyarpatti, Athoor, Dindigul	3130521	5.8	RE	re: 6/9p	6/9	WNL	re: Mod - Severe NPDR/CSME	210	396	110	100	46	RE 6/9	6/9	186	260		
43	green	Jose K.J	59	M	Kottayam	3253919	7.2	LE	le: 6/6p	6/6	early pscc	LE: severe npdr csme	151	472	100	100	64	le 6/6	6/6	148	302		
44	green	Narayanasamy	70	M	Sayanethal, Virudachalam	3257358	6.5	LE	le: 6/6p	6/6	pciol	LE: Mod npdr csme	225	407	100	100	61	le 6/6	6/6	210	390		
45	Yellow	Sandavaliyan	57	M	Vasantha Nagar, Madurai	3258577	7.4	RE	re: 6/6	6/6	lc	re: severe npdr csme	156	326	100	80	39	re 6/6	6/6	151	216		
46	green	Sandavaliyan	57	M	Vasantha Nagar, Madurai	3258577	7.4	LE	le: 6/6p	6/6	lc	LE: severe npdr csme	170	348	100	80	47	le 6/6	6/6	165	254		
47	yellow	Sivakumar	60	M	Aruppukottai, Virudhunagar	2987762	6.9	LE	le: 6/12p	6/12	pciol	LE: Mod npdr csme	320		120	100	46	le 6/9	6/9	236			
48	Yellow	Rajamani	60	F	K.K.Nagar, Madurai	2542384	8.4	LE	le: 6/24	6/24	pciol	LE: Mod npdr csme	215	326	80	100	24	le 6/12p	6/12	170	214		

49	Yellow	Rengaiah	58	M	Devapatti, Pudukottai	fh-1167618	5.4	LE	le: 6/12p	6/12	lc	LE: severe npdr csme	163	387	110	100	28	le 6/9	6/6	148	217		
50	green	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	8.3	LE	le: 6/9p	6/6	early imc	LE: Mod npdr csme	412	487	80	100	51	le 6/9	6/9	254	311		
51	Yellow	Vellaisamy	60	M	Kumaran Thirunagar, Dindigul	3169450	5.6	LE	le: 6/6p	6/6	wnl	LE: Mod npdr csme	227	455	90	100	32	le 6/6p	6/6	203	264		
52	green	Ravichandran	53	M	Philomina Nagar, Tanjore	2910825	6.2	LE	le: 6/6p	6/6	pciol	LE: severe npdr csme	187	380	150	80	22	le 6/6	6/6	166	224		
53	green	Chandra	58	F	Visaloor, Tanjore	3321651	7.4	RE	RE: 6/9	6/6	lens changes	re: severe npdr csme	160	295	160	100	34	le 6/9	6/9	164	270		
54	green	Ayyammal	51	F	Pillayar Koil street, sholavandan, V	3325541	5.6	RE	re:6/6	6/6	lc	re: Mod NPDR/CSME	286	380	120	100	70	RE 6/6	6/6	210	286		
55	green	Ayyammal	51	F	Pillayar Koil street, sholavandan, V	3325541	5.6	LE	le: 6/6	6/6	lc	LE: Mod npdr csme	490		110	100	60	le 6/9	6/9	360			
56	Yellow	Palanisamy	56	M	Dharapuram	2923207	6.0	RE	re: 6/6	6/6	wnl	RE: mod npdr csme	185	356	100	100	34	re 6/6	6/6	160	267		
57	green	Palanisamy	56	M	Dharapuram	2923207	6.0	LE	le: 6/6	6/6	wnl	LE: severe npdr csme	165	360	110	100	42	le 6/6	6/6	155	282		
58	green	Sivaranjini	57	M	Vellipatinam, Ramnad	3251256	8.5	LE	le: 6/12p	6/12	lens changes	LE: Mod npdr csme	184	426	120	100	75	le 6/9	6/9	178	284		
59	green	Muthuvel	60	M	Boopalathi Street, Rajapalayam	3237426	6.4	Re	re: 6/9	6/9	lens changes	re: Mod - Severe NPDR/CSME	144	378	120	80	60	RE 6/9	6/9	140	280		
60	green	Thilakavatthi	57	F	Pudur, Madurai	3248231	7.9	RE	re" 6/12p	6/12	WNL	re: severe npdr csme	311	446	110	100	64	RE: 6/9	6/9	236	312		
61	Yellow	Pandiaraj	43	M	Thirunagar, Dindigul	3269455	6.1	Re	Re 6/6p	6/6	WNL	RE: mod npdr csme	241	466	90	100	66	RE: 6/6	6/6	214	316		
62	Yellow	Narayanan	68	M	KK Nagar, Madurai	3256481	7.2	RE	Re: 6/12p	6/12	pciol	re: severe npdr csme	211	383	100	100	56	Re: 6/9	6/9	182	226		
63	green	Thasleema Begun	66	F	Kollam, Kerela	3168426	8.2	RE	Re: 6/18	6/18	pciol	re: severe npdr csme	362	480	100	100	42	Re: 6/9	6/9	264	344		
64	Yellow	Thasleema Begun	66	F	Kollam, Kerela	3168426	8.2	LE	Re: 6/18p	6/18	pciol	LE: severe npdr csme	341	466	90	100	38	Le: 6/12	6/12	216	260		
65	Yellow	Jeyakumar	46	M	Chockikulam, Madurai	3361412	5.8	RE	Re: 6/9p	6/6	WNL	RE: mod npdr csme	260	364	110	100	58	Re: 6/6	6/6	204	268		
66	green	Jeyakumar	46	M	Chockikulam, Madurai	3361412	5.8	LE	le: 6/9	6/9	WNL	LE: Mod npdr csme	221	312	120	100	64	Le: 6/6p	6/6	208	256		
67	green	K.S.Raju	58	M	Thillai Nagar, Trichy	3352489	7.1	LE	LE 6/24	6/24	lens changes	LE: Mod - Severe NPDR/CSME	382	471	110	100	38	LE: 6/18p	6/18	368	454	120, 100, 42	320u
68	green	Joseph K.J	68	M	Kottayam	3331624	7.5	RE	Re: 6/12p	6/12	pciol	re: severe npdr csme	318	512	110	100	54	Re: 6/9	6/6	246	390		
69	green	Joseph K.J	68	M	Kottayam	3331624	7.4	LE	LE: 6/18p	6/18	pciol	LE: severe npdr csme	346	490	120	100	62	LE: 6/12	6/12	260	344		
70	Yellow	Manickam	42	M	Vellakoil	3334296	6.9	RE	RE 6/18	6/18	WNL	re: severe npdr csme	324	542	100	100	48	re: 6/9	6/9	218	304		
71	Yellow	Manickam	42	M	Vellakoil	3334296	6.9	LE	le: 6/12p	6/12	WNL	LE: severe npdr csme	352	512	90	100	52	le: 6/6p	6/6	232	268		

MATER CHART

sno	group	name	age	sex	address	mrno	hba1c	study_eye	bcva	bcva_review	anterior_seg	fundus_diagnosis	baseoct_fovea	baseparafov	basepower	duration_ms	base_spots	va_4m	va_4mr	octfovea_4m	octpara_4m	parameters - repeat laser	Final outcome
1	Yellow	Velayudhan	58	M	Ezhachery, Kottayam	3087774	6.5	RE	R-6/9	6/9	early imc	R:Mod NPDR/CSME	496		100	100	25	RE 6/9	6/9	284			
2	Green	Savithri	55	F	Mattaparai, Dindigul	3098148	5.8	RE	R-6/18	6/18	early IMC	RE: Mod NPDR/CSME	243	507	90	100	55	RE 6/9	6/9	210	380		
3	Green	Savithri	55	F	Mattaparai, Dindigul	3098148	5.8	LE	LE - 6/9p	6/9	Early IMC	LE: Mod NPDR/CSME	235	455	90	100	32	LE 6/9	6/9	216	340		
4	Yellow	Dhanapackiam	52	F	Kambilampatti, Dindigul	3098234	6.8	RE	R-6/12P	6/12	lens changes	RE: Mod-Severe NPDR/CSME Hard Exudat	290		110	100	111	RE 6/9	6/9	171			
5	Green	Dhanapackiam	52	F	Kambilampatti, Dindigul	3098234	6.8	LE	L-6/6P	6/6	lens changes	LE: Mod NPDR/CSME	452		100	100	81	LE 6/6p	6/6	239			
6	Green	Shahul Hameed	62	M	Athirampatinam, Tanjore	3065765	7.2	RE	R - 6/18	6/18	IMC (PSCC)	RE- severe NPDR/CSME	370		100	100	39	RE 6/12	6/12	254			
7	Green	Manickam	70	M	Athoor, Dindigul	3078366	4.9	LE	le - 6/6p	6/6	wnl	L: ModNPDR/CSME	484		110	100	25	LE 6/6	6/6	420			
8	Yellow	Ramamoorthy MS	42	M	Aruuppukottai, Virudunagar	3100503	5.5	RE	RE 6/24	6/24	wnl	RE: severe npdr/csme		469	90	100	62	re 6/9	6/9	310			
9	Green	Ramamoorthy MS	42	M	Aruuppukottai, Virudunagar	3100503	5.5	LE	LE 6/24p	6/24	WNL	LE: severe npdr/csme		647	70	100	21	re 6/12	6/12	416			
10	Yellow	Salahudeen	60	M	Kollam, Kerela	3109224	6.4	RE	R 6/12p	6/12	wnl	Re: Mod NPDR CSME	423		120	100	38	RE 6/6p	6/6	226			
11	Green	Dharmarajan	65	M	Santhaipettai, Madurai	3168421	6.9	RE	Re: 6/24	6/24	lens changes	Re: Severe NPDR CSME with CME PVD	660		120	100	42	RE 6/18	6/18	480			
12	Yellow	Maragatham	40	F	Nethaji Road, Madurai	3106807	7.2	RE	Re 6/9p	6/9	Wnl	BE: Mod NPDR/CSME	188	399	160	100	11	RE 6/9	6/9	186	348		
13	Yellow	Suganthan	61	M	Varkala, Trivandrum	3133977	8.3	LE	LE 6/12	6/12	LC	LE: SEVERE NPDR/CSME	334		90	100	25	RE 6/9p	6/9	214			
14	Yellow	Subramnaniam	63	M	jayanthi Nagar, Madurai	3156654	7.9	RE	re 6/12p	6/12	wnl	BE: Mod NPDR/CSME	311	511	150	150	47	RE 6/9	6/9	220	344		
15	Yellow	Aysha Beevi	56	F	Thasiladar Nagar, Madurai	3167633	8.4	LE	LE 6/18	6/18	wnl	BE: Mod-Severe NPDR/CSME	309	482	120	100	24	LE 6/12	6/12	190	312		
16	Yellow	Premchand	50	M	Trivandrum	3190307	8.6	RE	re 6/9	6/9	wnl	BE: Mod-Severe NPDR/CSME	346		120	100	42	RE 6/9	6/9	391		110, 100, 51	270u
17	Yellow	Rabiyammul	72	F	Vilachery, Madurai	3193007	5.2	RE	re 6/24p	6/24	ns II	BE: Mod-Severe NPDR/CSME	296	400	140	120	41	RE 6/9	6/9	170	320		
18	Green	Kunhaysha	70	F	Vadakara, Kozhikode	3204785	6.4	LE	le 6/12	6/12	lc	BE: Severe NPDR/CSME	207	223	150	100	49	LE 6/9	6/9	180	112		
19	yellow	Indira	56	F	Kandalloor, Allepey	3208489	8.9	RE	re: 6/36	6/36	lc	BE: Severe NPDR/CSME	630		100	100	60	RE 6/18	6/18	415			
20	yellow	Indira	56	F	Kandalloor, Allepey	3208489	8.9	LE	le: 6/6p	6/6	lc	BE: Severe NPDR/CSME	188	328	100	100	34	LE 6/6	6/6	160	265		
21	yellow	Seshan	52	M	Simmakkal, Madurai	2906835	8.1	RE	re: 6/36p	6/36	early imc	BE: Severe NPDR/CSME	603		120	100	102	RE 6/36	6/36	384			
22	Green	Kalaiselvi	48	F	Annamalai Nagar, Tanjore	3143412	6.6	RE	re 6/18	6/18	imc	be: severe npdr/csme	319		100	100	29	RE 6/9	6/9	250			
23	Green	Kalaiselvi	48	F	Annamalai Nagar, Tanjore	3143412	6.6	LE	le 6/24	6/24	imc	be: severe npdr/csme	375		120	100	42	LE 6/18p	6/18	363			
24	Green	Seshan	52	M	Simmakkal, Madurai	2906835	8.1	LE	le: 6/12p	6/12	early imc	BE: Severe NPDR/CSME	415		130	100	94	LE 6/12	6/12	319			
25	Yellow	Abdul Wahab	65	M	Mudukulathur, Ramanathapuram	3128738	6.2	LE	le: 6/9	6/9	wnl	Be: Mod NPDR/CSME	248	453	140	100	46	LE 6/9p	6/9	186	290		
26	Yellow	Anthony P.C.	74	M	Ramakkal Medu P.O., Idukki, Kere	3214535	5.5	RE	re: 6/12p	6/12	early imc	Be:Severe NPDR/CSME	363		150	100	61	RE 6/12	6/12	250			
27	green	Karishma Begum	55	F	Sattankulam, Ramanathapuram	3184299	5.0	LE	le 6/12	6/12	lc	LE: Mod npdr csme	255	439	110	100	40	LE 6/9	6/9	210	294		
28	green	Kesari das	48	F	Thiruvarambu, Kanyakumari	3017220	8.0	RE	6/6p	6/6	lc	BE: Mod-Severe NPDR/CSME	241	391	140	100	36	le 6/6	6/6	160	265		
29	green	Ayyasamy	73	M	Valangaiman, Thiruvarur	3204341	8.6	RE	re: 6/18p	6/18	pciol	BE: Severe NPDR/CSME	625		110	100	34	re 6/9p	6/9	600		100, 100, 41	390u
30	Yellow	Paneer Selvam	58	M	Virudhunagar	3235638	6.4	RE	re 6/18	6/18	wnl	BE: Severe NPDR/CSME	410		90	100	42	Re 6/9p	6/9	240			
31	Yellow	Durairaj	42	M	Ramanathapuram	3128742	5.8	RE	re 6/6p	6/6	wnl	Re: Mod Npder with CSME	640		100	100	28	RE 6/6	6/6	218			
32	Yellow	Anthony P.C.	74	M	Ramakkal Medu P.O., Idukki, Kere	3214535	5.5	LE	le: 6/18	6/18	early imc	BE: Severe NPDR/CSME	420		150	100	52	LE 6/12	6/12	242			
33	Yellow	Chandra Devi	50	F	Mettupatti, Vadipatti	2218268	6.9	RE	re: 6/6p	6/6	wnl	Be: Mod NPDR/CSME	170	327	130	100	84	RE 6/6	6/6	168	244		
34	green	Lakshmanan	40	M	Thiruppalai, Madurai City	3235646	6.4	RE	re: 6/6	6/6	wnl	Be: Mod NPDR/CSME	199	383	100	100	53	RE 6/6	6/6	180	242		
35	Yellow	Chandra Devi	50	F	Mettupatti, Vadipatti	2218268	6.9	LE	le: 6/6p	6/6	wnl	Be: Mod NPDR/CSME	327	431	130	100	72	RE 6/6	6/6	217	311		
36	green	Ayyanar	52	M	Vellipattinam, Ramanathapuram	3240581	6.9	LE	le: 6/9	6/9	lens changes	LE: Mod npdr csme	184	440	120	100	78	le 6/6p	6/6	182	360		
37	green	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	7.1	RE	re:6/9	6/9	lc	re: Mod - Severe NPDR/CSME	327	519	120	100	48	Re 6/6	6/6	240	410		
38	yellow	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	7.1	RE	le:6/9p	6/9	lc	le: Mod - Severe NPDR/CSME	237	455	100	100	62	Re 6/6	6/6	186	270		
39	Yellow	Abdul Azeez	43	M	Malapuram, Kerala	3235639	7.4	RE	re: 6/6p	6/6	WNL	BE: Severe NPDR/CSME	245	480	120	100	51	Re 6/6	6/6	211	326		
40	Yellow	Ponnammal	66	F	Aruppukottai, Virudhunagar	3174419	5.6	RE	re: 6/12p	6/12	pciol	re: Mod - Severe NPDR/CSME	141	430	100	100	135	Re 6/9	6/9	136	260		
41	Yellow	Jose K.J	59	M	Kottayam	3253919	7.2	RE	re: 6/24	6/24	early pscc	re: Disc pallor/ Severe NPDR/CSME	163	435	80	100	80	RE 6/18	6/18	156	230		
42	Yellow	Ramalingam	63	M	Samiyarpatti, Athoor, Dindigul	3130521	5.8	RE	re: 6/9p	6/9	WNL	re: Mod - Severe NPDR/CSME	210	396	110	100	46	RE 6/9	6/9	186	260		
43	green	Jose K.J	59	M	Kottayam	3253919	7.2	LE	le: 6/6p	6/6	early pscc	LE: severe npdr csme	151	472	100	100	64	le 6/6	6/6	148	302		
44	green	Narayanasamy	70	M	Sayanethal, Virudachalam	3257358	6.5	LE	le: 6/6p	6/6	pciol	LE: Mod npdr csme	225	407	100	100	61	le 6/6	6/6	210	390		
45	Yellow	Sandavaliyan	57	M	Vasantha Nagar, Madurai	3258577	7.4	RE	re: 6/6	6/6	lc	re: severe npdr csme	156	326	100	80	39	re 6/6	6/6	151	216		
46	green	Sandavaliyan	57	M	Vasantha Nagar, Madurai	3258577	7.4	LE	le: 6/6p	6/6	lc	LE: severe npdr csme	170	348	100	80	47	le 6/6	6/6	165	254		
47	yellow	Sivakumar	60	M	Aruppukottai, Virudhunagar	2987762	6.9	LE	le: 6/12p	6/12	pciol	LE: Mod npdr csme	320		120	100	46	le 6/9	6/9	236			
48	Yellow	Rajamani	60	F	K.K.Nagar, Madurai	2542384	8.4	LE	le: 6/24	6/24	pciol	LE: Mod npdr csme	215	326	80	100	24	le 6/12p	6/12	170	214		

49	Yellow	Rengaiah	58	M	Devapatti, Pudukottai	fh-1167618	5.4	LE	le: 6/12p	6/12	lc	LE: severe npdr csme	163	387	110	100	28	le 6/9	6/6	148	217		
50	green	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	8.3	LE	le: 6/9p	6/6	early imc	LE: Mod npdr csme	412	487	80	100	51	le 6/9	6/9	254	311		
51	Yellow	Vellaisamy	60	M	Kumaran Thirunagar, Dindigul	3169450	5.6	LE	le: 6/6p	6/6	wnl	LE: Mod npdr csme	227	455	90	100	32	le 6/6p	6/6	203	264		
52	green	Ravichandran	53	M	Philomina Nagar, Tanjore	2910825	6.2	LE	le: 6/6p	6/6	pciol	LE: severe npdr csme	187	380	150	80	22	le 6/6	6/6	166	224		
53	green	Chandra	58	F	Visaloor, Tanjore	3321651	7.4	RE	RE: 6/9	6/6	lens changes	re: severe npdr csme	160	295	160	100	34	le 6/9	6/9	164	270		
54	green	Ayyammal	51	F	Pillayar Koil street, sholavandan, V	3325541	5.6	RE	re:6/6	6/6	lc	re: Mod NPDR/CSME	286	380	120	100	70	RE 6/6	6/6	210	286		
55	green	Ayyammal	51	F	Pillayar Koil street, sholavandan, V	3325541	5.6	LE	le: 6/6	6/6	lc	LE: Mod npdr csme	490		110	100	60	le 6/9	6/9	360			
56	Yellow	Palanisamy	56	M	Dharapuram	2923207	6.0	RE	re: 6/6	6/6	wnl	RE: mod npdr csme	185	356	100	100	34	re 6/6	6/6	160	267		
57	green	Palanisamy	56	M	Dharapuram	2923207	6.0	LE	le: 6/6	6/6	wnl	LE: severe npdr csme	165	360	110	100	42	le 6/6	6/6	155	282		
58	green	Sivaranjini	57	M	Vellipatinam, Ramnad	3251256	8.5	LE	le: 6/12p	6/12	lens changes	LE: Mod npdr csme	184	426	120	100	75	le 6/9	6/9	178	284		
59	green	Muthuvel	60	M	Boopalathi Street, Rajapalayam	3237426	6.4	Re	re: 6/9	6/9	lens changes	re: Mod - Severe NPDR/CSME	144	378	120	80	60	RE 6/9	6/9	140	280		
60	green	Thilakavatthi	57	F	Pudur, Madurai	3248231	7.9	RE	re" 6/12p	6/12	WNL	re: severe npdr csme	311	446	110	100	64	RE: 6/9	6/9	236	312		
61	Yellow	Pandiaraj	43	M	Thirunagar, Dindigul	3269455	6.1	Re	Re 6/6p	6/6	WNL	RE: mod npdr csme	241	466	90	100	66	RE: 6/6	6/6	214	316		
62	Yellow	Narayanan	68	M	KK Nagar, Madurai	3256481	7.2	RE	Re: 6/12p	6/12	pciol	re: severe npdr csme	211	383	100	100	56	Re: 6/9	6/9	182	226		
63	green	Thasleema Begun	66	F	Kollam, Kerela	3168426	8.2	RE	Re: 6/18	6/18	pciol	re: severe npdr csme	362	480	100	100	42	Re: 6/9	6/9	264	344		
64	Yellow	Thasleema Begun	66	F	Kollam, Kerela	3168426	8.2	LE	Re: 6/18p	6/18	pciol	LE: severe npdr csme	341	466	90	100	38	Le: 6/12	6/12	216	260		
65	Yellow	Jeyakumar	46	M	Chockikulam, Madurai	3361412	5.8	RE	Re: 6/9p	6/6	WNL	RE: mod npdr csme	260	364	110	100	58	Re: 6/6	6/6	204	268		
66	green	Jeyakumar	46	M	Chockikulam, Madurai	3361412	5.8	LE	le: 6/9	6/9	WNL	LE: Mod npdr csme	221	312	120	100	64	Le: 6/6p	6/6	208	256		
67	green	K.S.Raju	58	M	Thillai Nagar, Trichy	3352489	7.1	LE	LE 6/24	6/24	lens changes	LE: Mod - Severe NPDR/CSME	382	471	110	100	38	LE: 6/18p	6/18	368	454	120, 100, 42	320u
68	green	Joseph K.J	68	M	Kottayam	3331624	7.5	RE	Re: 6/12p	6/12	pciol	re: severe npdr csme	318	512	110	100	54	Re: 6/9	6/6	246	390		
69	green	Joseph K.J	68	M	Kottayam	3331624	7.4	LE	LE: 6/18p	6/18	pciol	LE: severe npdr csme	346	490	120	100	62	LE: 6/12	6/12	260	344		
70	Yellow	Manickam	42	M	Vellakoil	3334296	6.9	RE	RE 6/18	6/18	WNL	re: severe npdr csme	324	542	100	100	48	re: 6/9	6/9	218	304		
71	Yellow	Manickam	42	M	Vellakoil	3334296	6.9	LE	le: 6/12p	6/12	WNL	LE: severe npdr csme	352	512	90	100	52	le: 6/6p	6/6	232	268		

MATER CHART

sno	group	name	age	sex	address	mrno	hba1c	study_eye	bcva	bcva_review	anterior_seg	fundus_diagnosis	baseoct_fovea	baseparafov	basepower	duration_ms	base_spots	va_4m	va_4mr	octfovea_4m	octpara_4m	parameters - repeat laser	Final outcome
1	Yellow	Velayudhan	58	M	Ezhachery, Kottayam	3087774	6.5	RE	R-6/9	6/9	early imc	R:Mod NPDR/CSME	496		100	100	25	RE 6/9	6/9	284			
2	Green	Savithri	55	F	Mattaparai, Dindigul	3098148	5.8	RE	R-6/18	6/18	early IMC	RE: Mod NPDR/CSME	243	507	90	100	55	RE 6/9	6/9	210	380		
3	Green	Savithri	55	F	Mattaparai, Dindigul	3098148	5.8	LE	LE - 6/9p	6/9	Early IMC	LE: Mod NPDR/CSME	235	455	90	100	32	LE 6/9	6/9	216	340		
4	Yellow	Dhanapackiam	52	F	Kambilampatti, Dindigul	3098234	6.8	RE	R-6/12P	6/12	lens changes	RE: Mod-Severe NPDR/CSME Hard Exudat	290		110	100	111	RE 6/9	6/9	171			
5	Green	Dhanapackiam	52	F	Kambilampatti, Dindigul	3098234	6.8	LE	L-6/6P	6/6	lens changes	LE: Mod NPDR/CSME	452		100	100	81	LE 6/6p	6/6	239			
6	Green	Shahul Hameed	62	M	Athirampatinam, Tanjore	3065765	7.2	RE	R - 6/18	6/18	IMC (PSCC)	RE- severe NPDR/CSME	370		100	100	39	RE 6/12	6/12	254			
7	Green	Manickam	70	M	Athoor, Dindigul	3078366	4.9	LE	le - 6/6p	6/6	wnl	L: ModNPDR/CSME	484		110	100	25	LE 6/6	6/6	420			
8	Yellow	Ramamoorthy MS	42	M	Aruuppukottai, Virudunagar	3100503	5.5	RE	RE 6/24	6/24	wnl	RE: severe npdr/csme		469	90	100	62	re 6/9	6/9	310			
9	Green	Ramamoorthy MS	42	M	Aruuppukottai, Virudunagar	3100503	5.5	LE	LE 6/24p	6/24	WNL	LE: severe npdr/csme		647	70	100	21	re 6/12	6/12	416			
10	Yellow	Salahudeen	60	M	Kollam, Kerela	3109224	6.4	RE	R 6/12p	6/12	wnl	Re: Mod NPDR CSME	423		120	100	38	RE 6/6p	6/6	226			
11	Green	Dharmarajan	65	M	Santhaipettai, Madurai	3168421	6.9	RE	Re: 6/24	6/24	lens changes	Re: Severe NPDR CSME with CME PVD	660		120	100	42	RE 6/18	6/18	480			
12	Yellow	Maragatham	40	F	Nethaji Road, Madurai	3106807	7.2	RE	Re 6/9p	6/9	Wnl	BE: Mod NPDR/CSME	188	399	160	100	11	RE 6/9	6/9	186	348		
13	Yellow	Suganthan	61	M	Varkala, Trivandrum	3133977	8.3	LE	LE 6/12	6/12	LC	LE: SEVERE NPDR/CSME	334		90	100	25	RE 6/9p	6/9	214			
14	Yellow	Subramnaniam	63	M	jayanthi Nagar, Madurai	3156654	7.9	RE	re 6/12p	6/12	wnl	BE: Mod NPDR/CSME	311	511	150	150	47	RE 6/9	6/9	220	344		
15	Yellow	Aysha Beevi	56	F	Thasiladar Nagar, Madurai	3167633	8.4	LE	LE 6/18	6/18	wnl	BE: Mod-Severe NPDR/CSME	309	482	120	100	24	LE 6/12	6/12	190	312		
16	Yellow	Premchand	50	M	Trivandrum	3190307	8.6	RE	re 6/9	6/9	wnl	BE: Mod-Severe NPDR/CSME	346		120	100	42	RE 6/9	6/9	391		110, 100, 51	270u
17	Yellow	Rabiyammul	72	F	Vilachery, Madurai	3193007	5.2	RE	re 6/24p	6/24	ns II	BE: Mod-Severe NPDR/CSME	296	400	140	120	41	RE 6/9	6/9	170	320		
18	Green	Kunhaysha	70	F	Vadakara, Kozhikode	3204785	6.4	LE	le 6/12	6/12	lc	BE: Severe NPDR/CSME	207	223	150	100	49	LE 6/9	6/9	180	112		
19	yellow	Indira	56	F	Kandalloor, Allepey	3208489	8.9	RE	re: 6/36	6/36	lc	BE: Severe NPDR/CSME	630		100	100	60	RE 6/18	6/18	415			
20	yellow	Indira	56	F	Kandalloor, Allepey	3208489	8.9	LE	le: 6/6p	6/6	lc	BE: Severe NPDR/CSME	188	328	100	100	34	LE 6/6	6/6	160	265		
21	yellow	Seshan	52	M	Simmakkal, Madurai	2906835	8.1	RE	re: 6/36p	6/36	early imc	BE: Severe NPDR/CSME	603		120	100	102	RE 6/36	6/36	384			
22	Green	Kalaiselvi	48	F	Annamalai Nagar, Tanjore	3143412	6.6	RE	re 6/18	6/18	imc	be: severe npdr/csme	319		100	100	29	RE 6/9	6/9	250			
23	Green	Kalaiselvi	48	F	Annamalai Nagar, Tanjore	3143412	6.6	LE	le 6/24	6/24	imc	be: severe npdr/csme	375		120	100	42	LE 6/18p	6/18	363			
24	Green	Seshan	52	M	Simmakkal, Madurai	2906835	8.1	LE	le: 6/12p	6/12	early imc	BE: Severe NPDR/CSME	415		130	100	94	LE 6/12	6/12	319			
25	Yellow	Abdul Wahab	65	M	Mudukulathur, Ramanathapuram	3128738	6.2	LE	le: 6/9	6/9	wnl	Be: Mod NPDR/CSME	248	453	140	100	46	LE 6/9p	6/9	186	290		
26	Yellow	Anthony P.C.	74	M	Ramakkal Medu P.O., Idukki, Kere	3214535	5.5	RE	re: 6/12p	6/12	early imc	Be:Severe NPDR/CSME	363		150	100	61	RE 6/12	6/12	250			
27	green	Karishma Begum	55	F	Sattankulam, Ramanathapuram	3184299	5.0	LE	le 6/12	6/12	lc	LE: Mod npdr csme	255	439	110	100	40	LE 6/9	6/9	210	294		
28	green	Kesari das	48	F	Thiruvarambu, Kanyakumari	3017220	8.0	RE	6/6p	6/6	lc	BE: Mod-Severe NPDR/CSME	241	391	140	100	36	le 6/6	6/6	160	265		
29	green	Ayyasamy	73	M	Valangaiman, Thiruvarur	3204341	8.6	RE	re: 6/18p	6/18	pciol	BE: Severe NPDR/CSME	625		110	100	34	re 6/9p	6/9	600		100, 100, 41	390u
30	Yellow	Paneer Selvam	58	M	Virudhunagar	3235638	6.4	RE	re 6/18	6/18	wnl	BE: Severe NPDR/CSME	410		90	100	42	Re 6/9p	6/9	240			
31	Yellow	Durairaj	42	M	Ramanathapuram	3128742	5.8	RE	re 6/6p	6/6	wnl	Re: Mod Npder with CSME	640		100	100	28	RE 6/6	6/6	218			
32	Yellow	Anthony P.C.	74	M	Ramakkal Medu P.O., Idukki, Kere	3214535	5.5	LE	le: 6/18	6/18	early imc	BE: Severe NPDR/CSME	420		150	100	52	LE 6/12	6/12	242			
33	Yellow	Chandra Devi	50	F	Mettupatti, Vadipatti	2218268	6.9	RE	re: 6/6p	6/6	wnl	Be: Mod NPDR/CSME	170	327	130	100	84	RE 6/6	6/6	168	244		
34	green	Lakshmanan	40	M	Thiruppalai, Madurai City	3235646	6.4	RE	re: 6/6	6/6	wnl	Be: Mod NPDR/CSME	199	383	100	100	53	RE 6/6	6/6	180	242		
35	Yellow	Chandra Devi	50	F	Mettupatti, Vadipatti	2218268	6.9	LE	le: 6/6p	6/6	wnl	Be: Mod NPDR/CSME	327	431	130	100	72	RE 6/6	6/6	217	311		
36	green	Ayyanar	52	M	Vellipattinam, Ramanathapuram	3240581	6.9	LE	le: 6/9	6/9	lens changes	LE: Mod npdr csme	184	440	120	100	78	le 6/6p	6/6	182	360		
37	green	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	7.1	RE	re:6/9	6/9	lc	re: Mod - Severe NPDR/CSME	327	519	120	100	48	Re 6/6	6/6	240	410		
38	yellow	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	7.1	RE	le:6/9p	6/9	lc	le: Mod - Severe NPDR/CSME	237	455	100	100	62	Re 6/6	6/6	186	270		
39	Yellow	Abdul Azeez	43	M	Malapuram, Kerala	3235639	7.4	RE	re: 6/6p	6/6	WNL	BE: Severe NPDR/CSME	245	480	120	100	51	Re 6/6	6/6	211	326		
40	Yellow	Ponnammal	66	F	Aruppukottai, Virudhunagar	3174419	5.6	RE	re: 6/12p	6/12	pciol	re: Mod - Severe NPDR/CSME	141	430	100	100	135	Re 6/9	6/9	136	260		
41	Yellow	Jose K.J	59	M	Kottayam	3253919	7.2	RE	re: 6/24	6/24	early pscc	re: Disc pallor/ Severe NPDR/CSME	163	435	80	100	80	RE 6/18	6/18	156	230		
42	Yellow	Ramalingam	63	M	Samiyarpatti, Athoor, Dindigul	3130521	5.8	RE	re: 6/9p	6/9	WNL	re: Mod - Severe NPDR/CSME	210	396	110	100	46	RE 6/9	6/9	186	260		
43	green	Jose K.J	59	M	Kottayam	3253919	7.2	LE	le: 6/6p	6/6	early pscc	LE: severe npdr csme	151	472	100	100	64	le 6/6	6/6	148	302		
44	green	Narayanasamy	70	M	Sayanethal, Virudachalam	3257358	6.5	LE	le: 6/6p	6/6	pciol	LE: Mod npdr csme	225	407	100	100	61	le 6/6	6/6	210	390		
45	Yellow	Sandavaliyan	57	M	Vasantha Nagar, Madurai	3258577	7.4	RE	re: 6/6	6/6	lc	re: severe npdr csme	156	326	100	80	39	re 6/6	6/6	151	216		
46	green	Sandavaliyan	57	M	Vasantha Nagar, Madurai	3258577	7.4	LE	le: 6/6p	6/6	lc	LE: severe npdr csme	170	348	100	80	47	le 6/6	6/6	165	254		
47	yellow	Sivakumar	60	M	Aruppukottai, Virudhunagar	2987762	6.9	LE	le: 6/12p	6/12	pciol	LE: Mod npdr csme	320		120	100	46	le 6/9	6/9	236			
48	Yellow	Rajamani	60	F	K.K.Nagar, Madurai	2542384	8.4	LE	le: 6/24	6/24	pciol	LE: Mod npdr csme	215	326	80	100	24	le 6/12p	6/12	170	214		

49	Yellow	Rengaiah	58	M	Devapatti, Pudukottai	fh-1167618	5.4	LE	le: 6/12p	6/12	lc	LE: severe npdr csme	163	387	110	100	28	le 6/9	6/6	148	217		
50	green	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	8.3	LE	le: 6/9p	6/6	early imc	LE: Mod npdr csme	412	487	80	100	51	le 6/9	6/9	254	311		
51	Yellow	Vellaisamy	60	M	Kumaran Thirunagar, Dindigul	3169450	5.6	LE	le: 6/6p	6/6	wnl	LE: Mod npdr csme	227	455	90	100	32	le 6/6p	6/6	203	264		
52	green	Ravichandran	53	M	Philomina Nagar, Tanjore	2910825	6.2	LE	le: 6/6p	6/6	pciol	LE: severe npdr csme	187	380	150	80	22	le 6/6	6/6	166	224		
53	green	Chandra	58	F	Visaloor, Tanjore	3321651	7.4	RE	RE: 6/9	6/6	lens changes	re: severe npdr csme	160	295	160	100	34	le 6/9	6/9	164	270		
54	green	Ayyammal	51	F	Pillayar Koil street, sholavandan, V	3325541	5.6	RE	re:6/6	6/6	lc	re: Mod NPDR/CSME	286	380	120	100	70	RE 6/6	6/6	210	286		
55	green	Ayyammal	51	F	Pillayar Koil street, sholavandan, V	3325541	5.6	LE	le: 6/6	6/6	lc	LE: Mod npdr csme	490		110	100	60	le 6/9	6/9	360			
56	Yellow	Palanisamy	56	M	Dharapuram	2923207	6.0	RE	re: 6/6	6/6	wnl	RE: mod npdr csme	185	356	100	100	34	re 6/6	6/6	160	267		
57	green	Palanisamy	56	M	Dharapuram	2923207	6.0	LE	le: 6/6	6/6	wnl	LE: severe npdr csme	165	360	110	100	42	le 6/6	6/6	155	282		
58	green	Sivaranjini	57	M	Vellipatinam, Ramnad	3251256	8.5	LE	le: 6/12p	6/12	lens changes	LE: Mod npdr csme	184	426	120	100	75	le 6/9	6/9	178	284		
59	green	Muthuvel	60	M	Boopalathi Street, Rajapalayam	3237426	6.4	Re	re: 6/9	6/9	lens changes	re: Mod - Severe NPDR/CSME	144	378	120	80	60	RE 6/9	6/9	140	280		
60	green	Thilakavatthi	57	F	Pudur, Madurai	3248231	7.9	RE	re" 6/12p	6/12	WNL	re: severe npdr csme	311	446	110	100	64	RE: 6/9	6/9	236	312		
61	Yellow	Pandiaraj	43	M	Thirunagar, Dindigul	3269455	6.1	Re	Re 6/6p	6/6	WNL	RE: mod npdr csme	241	466	90	100	66	RE: 6/6	6/6	214	316		
62	Yellow	Narayanan	68	M	KK Nagar, Madurai	3256481	7.2	RE	Re: 6/12p	6/12	pciol	re: severe npdr csme	211	383	100	100	56	Re: 6/9	6/9	182	226		
63	green	Thasleema Begun	66	F	Kollam, Kerela	3168426	8.2	RE	Re: 6/18	6/18	pciol	re: severe npdr csme	362	480	100	100	42	Re: 6/9	6/9	264	344		
64	Yellow	Thasleema Begun	66	F	Kollam, Kerela	3168426	8.2	LE	Re: 6/18p	6/18	pciol	LE: severe npdr csme	341	466	90	100	38	Le: 6/12	6/12	216	260		
65	Yellow	Jeyakumar	46	M	Chockikulam, Madurai	3361412	5.8	RE	Re: 6/9p	6/6	WNL	RE: mod npdr csme	260	364	110	100	58	Re: 6/6	6/6	204	268		
66	green	Jeyakumar	46	M	Chockikulam, Madurai	3361412	5.8	LE	le: 6/9	6/9	WNL	LE: Mod npdr csme	221	312	120	100	64	Le: 6/6p	6/6	208	256		
67	green	K.S.Raju	58	M	Thillai Nagar, Trichy	3352489	7.1	LE	LE 6/24	6/24	lens changes	LE: Mod - Severe NPDR/CSME	382	471	110	100	38	LE: 6/18p	6/18	368	454	120, 100, 42	320u
68	green	Joseph K.J	68	M	Kottayam	3331624	7.5	RE	Re: 6/12p	6/12	pciol	re: severe npdr csme	318	512	110	100	54	Re: 6/9	6/6	246	390		
69	green	Joseph K.J	68	M	Kottayam	3331624	7.4	LE	LE: 6/18p	6/18	pciol	LE: severe npdr csme	346	490	120	100	62	LE: 6/12	6/12	260	344		
70	Yellow	Manickam	42	M	Vellakoil	3334296	6.9	RE	RE 6/18	6/18	WNL	re: severe npdr csme	324	542	100	100	48	re: 6/9	6/9	218	304		
71	Yellow	Manickam	42	M	Vellakoil	3334296	6.9	LE	le: 6/12p	6/12	WNL	LE: severe npdr csme	352	512	90	100	52	le: 6/6p	6/6	232	268		

MATER CHART

sno	group	name	age	sex	address	mrno	hba1c	study_eye	bcva	bcva_review	anterior_seg	fundus_diagnosis	baseoct_fovea	baseparafov	basepower	duration_ms	base_spots	va_4m	va_4mr	octfovea_4m	octpara_4m	parameters - repeat laser	Final outcome
1	Yellow	Velayudhan	58	M	Ezhachery, Kottayam	3087774	6.5	RE	R-6/9	6/9	early imc	R:Mod NPDR/CSME	496		100	100	25	RE 6/9	6/9	284			
2	Green	Savithri	55	F	Mattaparai, Dindigul	3098148	5.8	RE	R-6/18	6/18	early IMC	RE: Mod NPDR/CSME	243	507	90	100	55	RE 6/9	6/9	210	380		
3	Green	Savithri	55	F	Mattaparai, Dindigul	3098148	5.8	LE	LE - 6/9p	6/9	Early IMC	LE: Mod NPDR/CSME	235	455	90	100	32	LE 6/9	6/9	216	340		
4	Yellow	Dhanapackiam	52	F	Kambilampatti, Dindigul	3098234	6.8	RE	R-6/12P	6/12	lens changes	RE: Mod-Severe NPDR/CSME Hard Exudat	290		110	100	111	RE 6/9	6/9	171			
5	Green	Dhanapackiam	52	F	Kambilampatti, Dindigul	3098234	6.8	LE	L-6/6P	6/6	lens changes	LE: Mod NPDR/CSME	452		100	100	81	LE 6/6p	6/6	239			
6	Green	Shahul Hameed	62	M	Athirampatinam, Tanjore	3065765	7.2	RE	R - 6/18	6/18	IMC (PSCC)	RE- severe NPDR/CSME	370		100	100	39	RE 6/12	6/12	254			
7	Green	Manickam	70	M	Athoor, Dindigul	3078366	4.9	LE	le - 6/6p	6/6	wnl	L: ModNPDR/CSME	484		110	100	25	LE 6/6	6/6	420			
8	Yellow	Ramamoorthy MS	42	M	Aruuppukottai, Virudunagar	3100503	5.5	RE	RE 6/24	6/24	wnl	RE: severe npdr/csme		469	90	100	62	re 6/9	6/9	310			
9	Green	Ramamoorthy MS	42	M	Aruuppukottai, Virudunagar	3100503	5.5	LE	LE 6/24p	6/24	WNL	LE: severe npdr/csme		647	70	100	21	re 6/12	6/12	416			
10	Yellow	Salahudeen	60	M	Kollam, Kerela	3109224	6.4	RE	R 6/12p	6/12	wnl	Re: Mod NPDR CSME	423		120	100	38	RE 6/6p	6/6	226			
11	Green	Dharmarajan	65	M	Santhaipettai, Madurai	3168421	6.9	RE	Re: 6/24	6/24	lens changes	Re: Severe NPDR CSME with CME PVD	660		120	100	42	RE 6/18	6/18	480			
12	Yellow	Maragatham	40	F	Nethaji Road, Madurai	3106807	7.2	RE	Re 6/9p	6/9	Wnl	BE: Mod NPDR/CSME	188	399	160	100	11	RE 6/9	6/9	186	348		
13	Yellow	Suganthan	61	M	Varkala, Trivandrum	3133977	8.3	LE	LE 6/12	6/12	LC	LE: SEVERE NPDR/CSME	334		90	100	25	RE 6/9p	6/9	214			
14	Yellow	Subramnaniam	63	M	jayanthi Nagar, Madurai	3156654	7.9	RE	re 6/12p	6/12	wnl	BE: Mod NPDR/CSME	311	511	150	150	47	RE 6/9	6/9	220	344		
15	Yellow	Aysha Beevi	56	F	Thasiladar Nagar, Madurai	3167633	8.4	LE	LE 6/18	6/18	wnl	BE: Mod-Severe NPDR/CSME	309	482	120	100	24	LE 6/12	6/12	190	312		
16	Yellow	Premchand	50	M	Trivandrum	3190307	8.6	RE	re 6/9	6/9	wnl	BE: Mod-Severe NPDR/CSME	346		120	100	42	RE 6/9	6/9	391		110, 100, 51	270u
17	Yellow	Rabiyammul	72	F	Vilachery, Madurai	3193007	5.2	RE	re 6/24p	6/24	ns II	BE: Mod-Severe NPDR/CSME	296	400	140	120	41	RE 6/9	6/9	170	320		
18	Green	Kunhaysha	70	F	Vadakara, Kozhikode	3204785	6.4	LE	le 6/12	6/12	lc	BE: Severe NPDR/CSME	207	223	150	100	49	LE 6/9	6/9	180	112		
19	yellow	Indira	56	F	Kandalloor, Allepey	3208489	8.9	RE	re: 6/36	6/36	lc	BE: Severe NPDR/CSME	630		100	100	60	RE 6/18	6/18	415			
20	yellow	Indira	56	F	Kandalloor, Allepey	3208489	8.9	LE	le: 6/6p	6/6	lc	BE: Severe NPDR/CSME	188	328	100	100	34	LE 6/6	6/6	160	265		
21	yellow	Seshan	52	M	Simmakkal, Madurai	2906835	8.1	RE	re: 6/36p	6/36	early imc	BE: Severe NPDR/CSME	603		120	100	102	RE 6/36	6/36	384			
22	Green	Kalaiselvi	48	F	Annamalai Nagar, Tanjore	3143412	6.6	RE	re 6/18	6/18	imc	be: severe npdr/csme	319		100	100	29	RE 6/9	6/9	250			
23	Green	Kalaiselvi	48	F	Annamalai Nagar, Tanjore	3143412	6.6	LE	le 6/24	6/24	imc	be: severe npdr/csme	375		120	100	42	LE 6/18p	6/18	363			
24	Green	Seshan	52	M	Simmakkal, Madurai	2906835	8.1	LE	le: 6/12p	6/12	early imc	BE: Severe NPDR/CSME	415		130	100	94	LE 6/12	6/12	319			
25	Yellow	Abdul Wahab	65	M	Mudukulathur, Ramanathapuram	3128738	6.2	LE	le: 6/9	6/9	wnl	Be: Mod NPDR/CSME	248	453	140	100	46	LE 6/9p	6/9	186	290		
26	Yellow	Anthony P.C.	74	M	Ramakkal Medu P.O., Idukki, Kere	3214535	5.5	RE	re: 6/12p	6/12	early imc	Be:Severe NPDR/CSME	363		150	100	61	RE 6/12	6/12	250			
27	green	Karishma Begum	55	F	Sattankulam, Ramanathapuram	3184299	5.0	LE	le 6/12	6/12	lc	LE: Mod npdr csme	255	439	110	100	40	LE 6/9	6/9	210	294		
28	green	Kesari das	48	F	Thiruvarambu, Kanyakumari	3017220	8.0	RE	6/6p	6/6	lc	BE: Mod-Severe NPDR/CSME	241	391	140	100	36	le 6/6	6/6	160	265		
29	green	Ayyasamy	73	M	Valangaiman, Thiruvarur	3204341	8.6	RE	re: 6/18p	6/18	pciol	BE: Severe NPDR/CSME	625		110	100	34	re 6/9p	6/9	600		100, 100, 41	390u
30	Yellow	Paneer Selvam	58	M	Virudhunagar	3235638	6.4	RE	re 6/18	6/18	wnl	BE: Severe NPDR/CSME	410		90	100	42	Re 6/9p	6/9	240			
31	Yellow	Durairaj	42	M	Ramanathapuram	3128742	5.8	RE	re 6/6p	6/6	wnl	Re: Mod Npder with CSME	640		100	100	28	RE 6/6	6/6	218			
32	Yellow	Anthony P.C.	74	M	Ramakkal Medu P.O., Idukki, Kere	3214535	5.5	LE	le: 6/18	6/18	early imc	BE: Severe NPDR/CSME	420		150	100	52	LE 6/12	6/12	242			
33	Yellow	Chandra Devi	50	F	Mettupatti, Vadipatti	2218268	6.9	RE	re: 6/6p	6/6	wnl	Be: Mod NPDR/CSME	170	327	130	100	84	RE 6/6	6/6	168	244		
34	green	Lakshmanan	40	M	Thiruppalai, Madurai City	3235646	6.4	RE	re: 6/6	6/6	wnl	Be: Mod NPDR/CSME	199	383	100	100	53	RE 6/6	6/6	180	242		
35	Yellow	Chandra Devi	50	F	Mettupatti, Vadipatti	2218268	6.9	LE	le: 6/6p	6/6	wnl	Be: Mod NPDR/CSME	327	431	130	100	72	RE 6/6	6/6	217	311		
36	green	Ayyanar	52	M	Vellipattinam, Ramanathapuram	3240581	6.9	LE	le: 6/9	6/9	lens changes	LE: Mod npdr csme	184	440	120	100	78	le 6/6p	6/6	182	360		
37	green	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	7.1	RE	re:6/9	6/9	lc	re: Mod - Severe NPDR/CSME	327	519	120	100	48	Re 6/6	6/6	240	410		
38	yellow	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	7.1	RE	le:6/9p	6/9	lc	le: Mod - Severe NPDR/CSME	237	455	100	100	62	Re 6/6	6/6	186	270		
39	Yellow	Abdul Azeez	43	M	Malapuram, Kerala	3235639	7.4	RE	re: 6/6p	6/6	WNL	BE: Severe NPDR/CSME	245	480	120	100	51	Re 6/6	6/6	211	326		
40	Yellow	Ponnammal	66	F	Aruppukottai, Virudhunagar	3174419	5.6	RE	re: 6/12p	6/12	pciol	re: Mod - Severe NPDR/CSME	141	430	100	100	135	Re 6/9	6/9	136	260		
41	Yellow	Jose K.J	59	M	Kottayam	3253919	7.2	RE	re: 6/24	6/24	early pscc	re: Disc pallor/ Severe NPDR/CSME	163	435	80	100	80	RE 6/18	6/18	156	230		
42	Yellow	Ramalingam	63	M	Samiyarpatti, Athoor, Dindigul	3130521	5.8	RE	re: 6/9p	6/9	WNL	re: Mod - Severe NPDR/CSME	210	396	110	100	46	RE 6/9	6/9	186	260		
43	green	Jose K.J	59	M	Kottayam	3253919	7.2	LE	le: 6/6p	6/6	early pscc	LE: severe npdr csme	151	472	100	100	64	le 6/6	6/6	148	302		
44	green	Narayanasamy	70	M	Sayanethal, Virudachalam	3257358	6.5	LE	le: 6/6p	6/6	pciol	LE: Mod npdr csme	225	407	100	100	61	le 6/6	6/6	210	390		
45	Yellow	Sandavaliyan	57	M	Vasantha Nagar, Madurai	3258577	7.4	RE	re: 6/6	6/6	lc	re: severe npdr csme	156	326	100	80	39	re 6/6	6/6	151	216		
46	green	Sandavaliyan	57	M	Vasantha Nagar, Madurai	3258577	7.4	LE	le: 6/6p	6/6	lc	LE: severe npdr csme	170	348	100	80	47	le 6/6	6/6	165	254		
47	yellow	Sivakumar	60	M	Aruppukottai, Virudhunagar	2987762	6.9	LE	le: 6/12p	6/12	pciol	LE: Mod npdr csme	320		120	100	46	le 6/9	6/9	236			
48	Yellow	Rajamani	60	F	K.K.Nagar, Madurai	2542384	8.4	LE	le: 6/24	6/24	pciol	LE: Mod npdr csme	215	326	80	100	24	le 6/12p	6/12	170	214		

49	Yellow	Rengaiah	58	M	Devapatti, Pudukottai	fh-1167618	5.4	LE	le: 6/12p	6/12	lc	LE: severe npdr csme	163	387	110	100	28	le 6/9	6/6	148	217		
50	green	Karuppaiah	57	M	Sekkalai, Karaikudi	2571485	8.3	LE	le: 6/9p	6/6	early imc	LE: Mod npdr csme	412	487	80	100	51	le 6/9	6/9	254	311		
51	Yellow	Vellaisamy	60	M	Kumaran Thirunagar, Dindigul	3169450	5.6	LE	le: 6/6p	6/6	wnl	LE: Mod npdr csme	227	455	90	100	32	le 6/6p	6/6	203	264		
52	green	Ravichandran	53	M	Philomina Nagar, Tanjore	2910825	6.2	LE	le: 6/6p	6/6	pciol	LE: severe npdr csme	187	380	150	80	22	le 6/6	6/6	166	224		
53	green	Chandra	58	F	Visaloor, Tanjore	3321651	7.4	RE	RE: 6/9	6/6	lens changes	re: severe npdr csme	160	295	160	100	34	le 6/9	6/9	164	270		
54	green	Ayyammal	51	F	Pillayar Koil street, sholavandan, V	3325541	5.6	RE	re:6/6	6/6	lc	re: Mod NPDR/CSME	286	380	120	100	70	RE 6/6	6/6	210	286		
55	green	Ayyammal	51	F	Pillayar Koil street, sholavandan, V	3325541	5.6	LE	le: 6/6	6/6	lc	LE: Mod npdr csme	490		110	100	60	le 6/9	6/9	360			
56	Yellow	Palanisamy	56	M	Dharapuram	2923207	6.0	RE	re: 6/6	6/6	wnl	RE: mod npdr csme	185	356	100	100	34	re 6/6	6/6	160	267		
57	green	Palanisamy	56	M	Dharapuram	2923207	6.0	LE	le: 6/6	6/6	wnl	LE: severe npdr csme	165	360	110	100	42	le 6/6	6/6	155	282		
58	green	Sivaranjini	57	M	Vellipatinam, Ramnad	3251256	8.5	LE	le: 6/12p	6/12	lens changes	LE: Mod npdr csme	184	426	120	100	75	le 6/9	6/9	178	284		
59	green	Muthuvel	60	M	Boopalathi Street, Rajapalayam	3237426	6.4	Re	re: 6/9	6/9	lens changes	re: Mod - Severe NPDR/CSME	144	378	120	80	60	RE 6/9	6/9	140	280		
60	green	Thilakavatthi	57	F	Pudur, Madurai	3248231	7.9	RE	re" 6/12p	6/12	WNL	re: severe npdr csme	311	446	110	100	64	RE: 6/9	6/9	236	312		
61	Yellow	Pandiaraj	43	M	Thirunagar, Dindigul	3269455	6.1	Re	Re 6/6p	6/6	WNL	RE: mod npdr csme	241	466	90	100	66	RE: 6/6	6/6	214	316		
62	Yellow	Narayanan	68	M	KK Nagar, Madurai	3256481	7.2	RE	Re: 6/12p	6/12	pciol	re: severe npdr csme	211	383	100	100	56	Re: 6/9	6/9	182	226		
63	green	Thasleema Begun	66	F	Kollam, Kerela	3168426	8.2	RE	Re: 6/18	6/18	pciol	re: severe npdr csme	362	480	100	100	42	Re: 6/9	6/9	264	344		
64	Yellow	Thasleema Begun	66	F	Kollam, Kerela	3168426	8.2	LE	Re: 6/18p	6/18	pciol	LE: severe npdr csme	341	466	90	100	38	Le: 6/12	6/12	216	260		
65	Yellow	Jeyakumar	46	M	Chockikulam, Madurai	3361412	5.8	RE	Re: 6/9p	6/6	WNL	RE: mod npdr csme	260	364	110	100	58	Re: 6/6	6/6	204	268		
66	green	Jeyakumar	46	M	Chockikulam, Madurai	3361412	5.8	LE	le: 6/9	6/9	WNL	LE: Mod npdr csme	221	312	120	100	64	Le: 6/6p	6/6	208	256		
67	green	K.S.Raju	58	M	Thillai Nagar, Trichy	3352489	7.1	LE	LE 6/24	6/24	lens changes	LE: Mod - Severe NPDR/CSME	382	471	110	100	38	LE: 6/18p	6/18	368	454	120, 100, 42	320u
68	green	Joseph K.J	68	M	Kottayam	3331624	7.5	RE	Re: 6/12p	6/12	pciol	re: severe npdr csme	318	512	110	100	54	Re: 6/9	6/6	246	390		
69	green	Joseph K.J	68	M	Kottayam	3331624	7.4	LE	LE: 6/18p	6/18	pciol	LE: severe npdr csme	346	490	120	100	62	LE: 6/12	6/12	260	344		
70	Yellow	Manickam	42	M	Vellakoil	3334296	6.9	RE	RE 6/18	6/18	WNL	re: severe npdr csme	324	542	100	100	48	re: 6/9	6/9	218	304		
71	Yellow	Manickam	42	M	Vellakoil	3334296	6.9	LE	le: 6/12p	6/12	WNL	LE: severe npdr csme	352	512	90	100	52	le: 6/6p	6/6	232	268		